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Great Lakes Water Quality Initiative Criteria Documents for the Protection of Wildlife (PROPOSED)

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Great Lakes Water Quality Initiative Criteria Documents for the Protection of Wildlife (PROPOSED) DDT; Mercury; 2,3,7,8-TCDD; PCBs

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Office of Science and Technology
Office of Water
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Washington, D.C. 20460

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Authors

Steven Bradbury
U.S. EPA, Environmental Research Laboratory
Duluth, MN

Cynthia Nolt
U.S. EPA, Office of Science and Technology,
Office of Water, Washington, DC

Beth Goodman
Wisconsin Department of Natural Resources
Madison, WI

Kenneth Stromborg
U.S. Fish and Wildlife Service
Green Bay, WI

John Sullivan
Wisconsin Department of Natural Resources
Madison, WI

Technical Support

Patrick Fitzsimmons
ASCI, Duluth, MN

Elizabeth Mooney
SAIC, Fairfax, VA

Isaac Diwan
SAIC, Fairfax, VA

Jeffrey Peterson
ManTech Environmental
Corvallis, OR

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Contents

CHAPTER 1

Tier I Wildlife Criteria for *p,p'*- Dichlorodiphenyltrichloroethane (DDT) and Metabolites

I. Literature Review 1-1

II. Calculation of Mammalian Wildlife Value 1-1
- i. Acute Toxicity Studies 1-1
- ii. Chronic Toxicity Studies 1-2
- iii. Mammalian Wildlife Value Calculation 1-3
- iv. Sensitivity Analysis for Mammalian Wildlife Value 1-4

III. Calculation of Avian Wildlife Value 1-5
- i. Acute Toxicity Studies 1-5
- ii. Chronic Toxicity Studies 1-6
- iii. Avian Wildlife Value Calculation 1-7
- iv. Sensitivity Analysis for Avian Wildlife Value 1-9

IV. Great Lakes Wildlife Criterion 1-9
- i. Discussion of Uncertainties 1-10

V. References 1-10

CHAPTER 2

Tier I Wildlife Criteria for Mercury (Including Methylmercury)

I. Literature Review 2-1

II. Calculation of Mammalian Wildlife Value 2-1
- i. Acute Toxicity Studies 2-1
- ii. Chronic Toxicity Studies 2-1
- iii. Mammalian Wildlife Value Calculation 2-2
- iv. Sensitivity Analysis for Mammalian Wildlife Value 2-3

III. Calculation of Avian Wildlife Value 2-4
- i. Acute Toxicity Studies 2-4
- ii. Chronic Toxicity Studies 2-5
- iii. Avian Wildlife Value Calculation 2-7
- iv Sensitivity Analysis for Avian Wildlife Value 2-8

IV. Great Lakes Wildlife Criterion 2-9
- i. Discussion of Uncertainties 2-9

V. References 2-9

Contents

CHAPTER 1
Tier I Wildlife Criteria for *p,p'*- Dichlorodiphenyltrichloroethane (DDT) and Metabolites

I. Literature Review 1-1

II. Calculation of Mammalian Wildlife Value 1-1
- i. Acute Toxicity Studies 1-1
- ii. Chronic Toxicity Studies 1-2
- iii. Mammalian Wildlife Value Calculation 1-3
- iv. Sensitivity Analysis for Mammalian Wildlife Value 1-4

III. Calculation of Avian Wildlife Value 1-5
- i. Acute Toxicity Studies 1-5
- ii. Chronic Toxicity Studies 1-6
- iii. Avian Wildlife Value Calculation 1-7
- iv. Sensitivity Analysis for Avian Wildlife Value 1-9

IV. Great Lakes Wildlife Criterion 1-9
- i. Discussion of Uncertainties 1-10

V. References 1-10

CHAPTER 2
Tier I Wildlife Criteria for Mercury (Including Methylmercury)

I. Literature Review 2-1

II. Calculation of Mammalian Wildlife Value 2-1
- i. Acute Toxicity Studies 2-1
- ii. Chronic Toxicity Studies 2-1
- iii. Mammalian Wildlife Value Calculation 2-2
- iv. Sensitivity Analysis for Mammalian Wildlife Value 2-3

III. Calculation of Avian Wildlife Value 2-4
- i. Acute Toxicity Studies 2-4
- ii. Chronic Toxicity Studies 2-5
- iii. Avian Wildlife Value Calculation 2-7
- iv Sensitivity Analysis for Avian Wildlife Value 2-8

IV. Great Lakes Wildlife Criterion 2-9
- i. Discussion of Uncertainties 2-9

V. References 2-9

CHAPTER 1
Tier I Wildlife Criteria for *p,p'*-Dichlorodiphenyltrichloroethane (DDT) and Metabolites

Contents

I. Literature Review 1-1

II. Calculation of Mammalian Wildlife Value 1-1

i. Acute Toxicity Studies 1-1

ii. Chronic Toxicity Studies 1-2

iii. Mammalian Wildlife Value Calculation 1-3

iv. Sensitivity Analysis for Mammalian Wildlife Value 1-4

III. Calculation of Avian Wildlife Value 1-5

i. Acute Toxicity Studies 1-5

ii. Chronic Toxicity Studies 1-6

iii. Avian Wildlife Value Calculation 1-7

iv. Sensitivity Analysis for Avian Wildlife Value 1-9

IV. Great Lakes Wildlife Criterion 1-9

i. Discussion of Uncertainties 1-10

V. References 1-10

Tier I Wildlife Criteria for *p,p'*-Dichlorodiphenyltrichloroethane (DDT) and Metabolites

I. Literature Review

A review of mammalian and avian toxicity data for *p,p'*-dichlorodiphenyl-trichloroethane (DDT) and its metabolites was based on literature received through computer-based (CAS and BIOSES) as well as manual searches. A total of 36 references were screened for dose-response data. The majority of those references consisted of studies on avian species. Those references which were reviewed in detail, specifically those that contain dose-response data, are cited in Section V.

II. Calculation of Mammalian Wildlife Value

i. Acute Toxicity Studies

According to the RTECS database (NIOSH, 1992), the oral LD_{50} values for DDT range from 87 mg/kg for the rat to more than 5000 mg/kg for the hamster (See Table 1-1). LD_{50} values for DDT from other exposure routes range from 0.91-1931 mg/kg (NIOSH, 1992).

Table 1-1. Mammalian Acute Toxicity Values

Route	Species	LD_{50} (mg/kg)
oral	rat	87
oral	rat	152.3[a]
oral	mouse	135
oral	dog	150
oral	monkey	200
oral	cat	250
oral	rabbit	250
oral	guinea pig	150
oral	hamster	> 5000
dermal	rat	1931
dermal	rabbit	300
dermal	guinea pig	1000
i.p.	rat	0.91
i.p.	mouse	32

Table 1-1. Mammalian Acute Toxicity Values (Cont.)

Route	Species	LD_{50} (mg/kg)
s.c.	rat	1500
s.c.	rabbit	250
s.c.	guinea pig	900
i.v.	rat	68
i.v.	mouse	6.85
i.v.	dog	150
i.v.	monkey	50
i.v.	cat	40
i.v.	rabbit	50
unreported	rat	300
unreported	mammal	200

Source: NIOSH (1992), except for * Mijavila et al. (1981).

ii. Chronic Toxicity Studies

No suitable subchronic or chronic studies were found for mammalian wildlife in which dose-response data was reported. Gilbert (1969) did examine the toxicity of DDE to mink, although no dose-response data could be developed because exposures to DDT were intermittent and total DDT intake was unquantifiable, due to the experimental protocol that was used. Gilbert (1969) fed 10 male and 10 female mink a contaminated fish ration containing 0.58 ppm DDE. Three male and 2 female mink died within 20 days. The remainder of the experimental group was then maintained on a control ration, and intermittently on contaminated feed, for two different periods lasting up to 47 days. DDE residues were found to be greatest in the liver and brain tissues of the experimental animals and the spleen, adrenal glands, and testes were heavier in experimental animals than in controls. The whelping rate among the experimental animals was approximately half that of controls, and the average number of live kits 24 hours after birth was significantly reduced among the experimental females. Average *in utero* loss and average total loss of kits was also greater among the experimental group than among the controls.

Chronic and subchronic studies of the toxicity of DDT to mammals have been conducted using typical laboratory animals. In one subchronic study (Mitjavila et al., 1981), OFA Sprague Dawley rats (male, 32 per group) were administered *p,p'* DDT in an oil vehicle by gavage at a dose of 14.5 mg/kg/day for up to 52 days. Liver weight in the treated group was 20 percent greater than in the control group due to cellular hypertrophy induced by the DDT, and the level of total lipids was 30 percent less in the treated group than in the controls.

In another subchronic rat-feeding study (Laug et al., 1950), weanling rats (15/sex/group) were fed commercial-grade DDT (81 percent *p,p'*- DDT, 19 percent *o,p'*-DDT) at levels of 0, 1, 5, 10 or 50 ppm for 1-27 weeks. The critical effect was liver toxicity, demonstrated as relatively mild dose-dependent histopathologic changes in hepatocytes at doses of 5 ppm or higher. These changes included hepatocellular hypertrophy, increased cytoplasmic oxyphillia, and peripheral basophilic cytoplasmic granules. The NOAEL was 1 ppm. Based on a rat body weight of 0.20 kg and food ingestion rate of 0.01 kg/d (i.e., 5 percent body weight per day) (NIOSH, 1992), the LOAEL for liver effects was 0.25 mg/kg/day (5 ppm) and the NOAEL for liver effects was 0.05 mg/kg/day.

Both the studies by Mitjavila et al. (1981) and Laug et al. (1950) would not be acceptable for derivation of a mammalian wildlife value (WV) because of study duration, requirement for dose-response data and/or because the toxicity endpoint is not an acceptable endpoint as defined in the wildlife criteria methodology provided in Appendix D to 40 CFR 132. (For the purpose of wildlife criteria derivation, an acceptable subchronic or chronic endpoint is one that affects organismal growth or viability, or reproductive or developmental success or any other endpoint which is, or is directly related to, parameters that influence population dynamics.) These studies are presented here to provide relative perspective for doses at which toxicological impacts occur.

In a 2-year reproduction study (Fitzhugh, 1948), rats were provided a diet that contained 0, 10, 50, 100, and 600 ppm DDT. The number of litters, number of live young at birth, average weight at birth, and the number of young surviving through the weaning period were quantified. The number of litters, number of living young at birth, and average weight at birth did not appear to differ with dosage level. At a concentration of 50 ppm DDT, the number of weanling rats was reduced by approximately 20 percent. The NOAEL was 10 ppm DDT since no effect was observed at that level. Based on a rat body weight of 0.20 kg and a food ingestion rate of 0.01 kg/d (NIOSH, 1992), the LOAEL derived from this study was 2.5 mg/kg/day (50 ppm) and the NOAEL derived for calculation of a mammalian wildlife value was 0.50 mg/kg/day.

The results of the mammalian studies described above are summarized in Table 1-2.

Table 1-2. Summary of Chronic Mammalian Studies

Species	LOAEL (mg/kg/day)	NOAEL (mg/kg/day)	Toxic Effect Observed	Reference
Mink	n/a	n/a	Reproductive	Gilbert, 1969
Rat	14.5		Liver toxicity	Mitjavila et al., 1981
Rat	0.25	0.05	Liver toxicity	Laug et al., 1950
Rat	2.5	0.5	Reproductive	Fitzhugh et al., 1948

The study by Fitzhugh (1948) was selected for developing Tier I mammalian wildlife values because the Fitzhugh (1948) study consists of repeated oral exposures for over a 90-day period, and reports observed reproductive effects from chronic exposures. Therefore, this study fulfills the requirements for an appropriate study for wildlife criteria development as described in Appendix D to 40 CFR 132. The LOAEL for reproductive effects reported in Fitzhugh (1948) was 2.5 mg/kg/day (50 ppm) and the NOAEL was 0.5 mg/kg/day (10 ppm).

iii. Mammalian Wildlife Value Calculation

In calculating a Tier I wildlife value, a species sensitivity factor (SSF) of 1 to 0.01 is recommended in Appendix D to 40 CFR 132 to accommodate differences in interspecies toxicity. Because of the paucity of subchronic or chronic mammalian toxicity studies assessing the toxicity of DDT or its metabolites, a SSF of 0.1 is used to reflect the uncertainty in extrapolating toxicity data from the rat to the mink and river otter.

DDT bioaccumulation factor (BAF) values for Trophic Level 3 and Trophic Level 4 were derived based on the Great Lakes Water Quality Initiative procedure to determine a bioaccumulation factor presented in Appendix B to 40 CFR 132.

Input parameters for the wildlife equation are presented below.

NOAEL (mammalian)	=	0.5 mg/kg body weight/day
BAF_3 (Trophic Level 3)	=	1,000,000 ℓ/kg body weight
BAF_4 (Trophic Level 4)	=	3,000,000 ℓ/kg body weight
SSF	=	0.1 (mink and otter)

Body weights (Wt_A), ingestion rates (F_A), and drinking rates (W_A) for mink and river otter are presented in Table D-2 of Appendix D to 40 CFR 132 and shown below.

Wt_A (mink)	=	1.0 kg
Wt_A (otter)	=	8.0 kg
F_A (mink)	=	0.15 kg/day
F_A (otter)	=	0.9 kg/day
W_A (mink)	=	0.099 ℓ/day
W_A (otter)	=	0.64 ℓ/day

The wildlife equations and calculations of mammalian wildlife values are presented below:

$$WV\ (mink) = \frac{[NOAEL \times SSF] \times Wt_{A(mink)}}{W_{A(mink)} + [(1.0)(F_{A(mink)} \times BAF_3)]}$$

$$WV\ (mink) = \frac{(0.5\ mg/kg/d \times 0.1)\ 1.0\ kg}{0.099\ \ell/d + [(1.0)(.15\ kg/d \times 1{,}000{,}000\ \ell/kg)]}$$

$$WV\ (mink) = 333\ pg/\ell$$

$$WV\ (otter) = \frac{[NOAEL \times SSF] \times Wt_{A(otter)}}{W_{A(otter)} + [(0.5)(F_{A(otter)} \times BAF_3) + (0.5)(F_{A(otter)} \times BAF_4)]}.$$

$$WV\ (otter) = \frac{(0.5\ mg/kg/d \times 0.1)\ 8.0\ kg}{0.64\ \ell/d + [(0.5)(0.90\ kg/d \times 1{,}000{,}000\ \ell/kg) + (0.5)(0.90\ kg/d \times 3{,}000{,}000\ \ell/kg)]}$$

$$WV\ (otter) = 222\ pg/\ell$$

The geometric mean of these two mammalian wildlife values results in

$$WV\ (mammalian) = e^{[(\ln WV(mink) + \ln WV(otter))/2)}$$

$$WV\ (mammalian) = e^{[(\ln 333\ pg/\ell + \ln 222\ pg/\ell)/2)}$$

$$WV\ (mammalian) = 270\ pg/\ell.$$

iv. Sensitivity Analysis for Mammalian Wildlife Value

The values of the various parameters used to derive the mammalian wildlife value presented above represent the most reasonable assumptions. The purpose of this section is to illustrate the significance of these assumptions and the variability in the mammalian wildlife value if other assumptions are made for the values of the various parameters from which the mammalian wildlife value is derived. The intent of this section is to let the risk manager know, as much as

possible, the influence on the magnitude of the mammalian wildlife value of the assumptions made in its derivation.

In developing a mammalian wildlife value for DDT, reproductive effects of toxicity were judged to be of importance to protect wildlife populations. Toxic effects of DDT on the liver were observed at concentrations lower than the NOAEL for reproductive effects. The NOAEL determined in the Laug et al. (1950) rat study suggests that toxic effects on the liver can occur at DDT doses that are as much as 10 times lower than the NOAEL for reproduction of 0.5 mg/kg/day determined by Fitzhugh (1948) and used to calculate the mammalian wildlife value. Although the methodology does not allow for basing a wildlife criterion on a liver toxicity endpoint, if one used the NOAEL from the Laug et al. (1950) study, the mammalian wildlife value would be 27 pg/ℓ rather than 270 pg/ℓ.

In estimating the hazards of DDT to mammalian wildlife, a SSF of 0.1 was used to reflect the uncertainty in extrapolating toxicity data from the rat to mink and river otter. Based on the lack of mammalian chronic toxicity data, the use of such a factor seems reasonable. Oral acute toxicity values (Table 1-1) show a wide variability in sensitivity within a species, but they also show that the rat is among the most sensitive of the mammalian species tested for the acute effects of DDT. This may justify the use of an alternate and less conservative SSF of 0.3--the geometric mean of 1 and 0.1. If an intermediate SSF of 0.3 were used with the NOAEL determined in the Fitzhugh (1948) study, the mammalian wildlife value would be 820 pg/ℓ instead of 270 pg/ℓ.

In deriving the DDT mammalian wildlife value, it was assumed that 100 percent of the mink diet was comprised of fish, although this may not necessarily be the case. This assumption may lead to an overestimate of DDT exposure for mink that are not primarily feeding on fish and aquatic invertebrates. As indicated in the Technical Support Document for Wildlife Criteria (Appendix to the Preamble to 40 CFR 132), the fish content of a mink diet can vary from less than 50 percent to the 100 percent assumed in the mink wildlife value derivation presented above. If it were assumed only 50 percent of a mink's diet was from aquatic resources and the remaining 50 percent of the diet was uncontaminated, the estimated DDT exposure would be reduced by a factor of 2. The resulting wildlife value for the mink would be 670 pg/ℓ, and the mammalian wildlife value would be 380 pg/ℓ, rather than the mammalian wildlife value of 270 pg/ℓ.

III. Calculation of Avian Wildlife Value

i. Acute Toxicity Studies

Long-term exposure of birds to DDT has been demonstrated to result in eggshell thinning in several species; however, the acute toxicity of DDT has not been well established. The RTECS database (NIOSH, 1992) listed the oral LD_{50} value for chickens (*Gallus*) as 300 mg/kg. Bernard (1963) observed tremors within 7 days in robins (*Turdus migratorius*) ingesting feed contaminated with 300 mg/kg DDT. Stickel et al. (1966) reported the oral DDT LC_{50} for bald eagles (*Haliaetus leucocephalus*) as 80 ppm following dietary exposure for 3-4 months. For the clapper rails (*Rallus longirostris*), the DDT oral LC_{50} value was 1612 ppm for males and 1896 ppm for females. The LC_{50} value for juvenile (2 to 3 weeks old) ring-necked pheasant (*Phasianus colchicus*) was 311 ppm, while the value for juvenile mallard ducks (*Anas platyrhynchos*) was found to be 1869 ppm (Van Veltzen and Kreitzer, 1975).

LC_{50} values for DDT concentrations in brain tissue have also been determined for avian species. The geometric mean brain DDT residue LC_{50} values ranged from 23 ppm wet weight for the blue jay (*Cyanocitta cristata*) to 109 ppm wet weight for the cardinal (*Richmondena cardinalis*) (Van Veltzen and Kreitzer, 1975). Stickel et al. (1984) established that 300-400 ppm DDE wet weight in brain tissue caused death in grackles (*Quiscalus guiscula*), red-winged blackbirds (*Agelaius phoeniceus*), brown-headed cowbirds (*Molothrus ater*) and starlings (*Sturnus vulgaris*). DDE residues in brains of two kestrels (*Falco sparverius*) that died following 14 months of exposure to 2.8 ppm dietary DDE (wet weight, or 10 ppm dry weight) were 212.5 and 301.1 ppm wet weight (Porter and Wiemeyer, 1972).

ii. Chronic Toxicity Studies

The toxicity of DDT has been documented in a number of avian species including the mallard (Kolaja, 1977; Heath et al., 1969; Davison and Sell, 1974), kestrels (Peakall et al., 1973), and brown pelicans (*Pelecanus occidentalis*) (Anderson et al., 1975).

Numerous studies of DDT and/or DDE ingestion by mallard ducks at levels ranging from 10 to 40 ppm in feed for a period ranging from 5 weeks prior to egg laying and through two years have demonstrated significant reduction in eggshell thickness (Haegele and Hudson, 1974; Longcore and Samson, 1973; Davison and Sell, 1973; Risebrough and Anderson, 1975; Kolaja and Hinton, 1977).

Davison and Sell (1974) exposed female mallards to technical grade DDT and pure *p,p'*-DDT at 0, 2, 20, and 200 ppm in the diet and assessed effects on eggshell thickness. Significant reduction in eggshell thickness was observed at 20 ppm (the LOAEL), and the NOAEL was 2 ppm for eggshell thickness. Lethality was observed at 200 ppm dietary DDT. Using a mallard body weight of 1 kg (Delnicki and Reinecke, 1986), and a feeding rate of 0.0582 kg/d determined using the allometric relationship provided in Appendix D to 40 CFR 132, a LOAEL value of 1.16 mg/kg/day (20 ppm) and a NOAEL of 0.116 mg/kg/day (2 ppm) can be estimated for effects on eggshell thickness.

Kolaja (1977) quantified effects of dietary DDT and DDE on mallard duck eggshell thickness and weight. Birds were exposed to dietary DDT and DDE at 0, 10 and 50 ppm. Eggshell thickness and weight were significantly reduced at both dose levels for either DDT or DDE. Using the mallard body weight and ingestion rate presented above, the LOAEL determined in this study is 2.91 mg/kg/day for eggshell thickness and weight.

Heath et al. (1969) exposed mallard ducks to dietary DDT, DDE, and DDD for 2 years and assessed reproductive success and eggshell thinning. Ducks were exposed to dietary DDE and DDD in commercial feed at 10 and 40 ppm and DDT at 2.5, 10, and 40 or 25 ppm (the higher concentration was reduced after breeders died). Endpoints evaluated were percent cracked eggs, embryo mortality, hatchling survivability, and number of ducklings per hen. DDE severely impaired reproductive success at both dose levels, and duckling production per hen was reduced by 50 to 75 percent. The DDE LOAEL for reproductive success obtained from this study was 10 ppm, or 0.58 mg/kg/day calculated using the default body weight and feed ingestion rate presented previously. Heath et al. (1969) also reported that DDD impaired reproductive success, but less severely than did DDE. DDT in the diet at concentrations of 2.5 and 10 ppm did not have measurable effects on reproduction. Therefore, the LOAEL for DDT in the diet of mallard ducks based on reproductive success is 1.45 mg/kg/day (25 ppm) and the NOAEL is 0.58 mg/kg/day (10 ppm).

Peakall et al. (1973) exposed American kestrels to 3, 6, and 10 ppm DDE in the diet and measured eggshell thickness, breaking strength, and permeability. Significant effects on each of these endpoints were observed at the lowest dietary concentration. Using a default kestrel body weight of 100 g (Bloom, 1973), and an ingestion rate derived from the allometric relationship presented in the Appendix D to 40 CFR 132, the LOAEL determined for DDE in this study is 0.39 mg/kg/day (3 ppm).

Alsop (1972) compared red-winged blackbird eggs collected during two successive seasons in an area ranging from Tennessee to Florida to pre-DDT eggs from museum collections. Post-DDT eggs were significantly thinner than those in the museum collections. However, since no measurements of DDT levels were performed on either the field sampled eggs or the museum collection, attributing the effect to DDT alone is speculative.

Anderson et al. (1975) studied the reproductive success of brown pelicans off the coast of southern California for the years of 1969 through 1974. Concentrations of DDT and metabolites in anchovies, the major food source of this pelican colony, and pelican eggs were also measured during the course of this investigation. Over the five years, combined concentrations of DDT, DDD, and DDE in the food source steadily declined from 4.27 ppm (wet weight) in 1969 to 0.15 ppm in 1974. At 0.15 ppm total DDT and metabolites in the food source, the fledgling rate was

30 percent below the estimated rate necessary to maintain a stationary population. Based on the results of this study, a LOAEL of 0.15 ppm total DDT can be inferred for reproductive success. Using a pelican body weight of 3.5 kg (Dunning, 1984), and the allometric equations presented in Appendix D to 40 CFR 132, the calculated food ingestion rate for pelicans is 0.131 kg/day (dry weight). Since the DDT bioaccumulation factor for the pelican's food source is provided in terms of wet weight, the calculated dry weight food ingestion rate is converted to a wet weight food ingestion rate by multiplying by 5 (U.S. EPA, 1980). This results in an intake rate of 0.66 kg/d. Multiplying the LOAEL (0.15 ppm) by the food ingestion rate and dividing by the pelican body weight gives a LOAEL of 2.82×10^{-2} mg/kg/day for reproductive success. The results of the studies described above are summarized in Table 1-3.

The Anderson et al. (1975) study with brown pelicans was judged most appropriate for avian wildlife value development because it consists of a peer-reviewed field study of a wildlife species that provides a chemical-specific dose-response curve for reproductive success. According to the methodology presented in Appendix D to 40 CFR 132, a study of this type takes precedence over other studies in the development of a Tier I criterion.

Table 1-3. Summary of Chronic Avian Studies

Species	LOAEL (mg/kg/day)	NOAEL (mg/kg/day)	Toxic Effect Observed	Reference
Mallard	1.16	0.116	Eggshell thinning	Davison and Sell, 1975
Mallard	2.91		Eggshell thinning	Kolaja, 1977
Mallard	0.58		For DDE : Reproductive effects (Embryo mortality, cracked eggs)	Heath et al., 1969
Mallard	1.45	0.58	For DDT: Reproductive effects	Heath et al., 1969
Kestrel	0.39		Eggshell thinning	Peakall et al., 1973
Pelican	0.028		Reproductive effects	Anderson et al., 1975

iii. Avian Wildlife Value Calculation

The LOAEL is divided by a LOAEL to NOAEL uncertainty factor of 10, resulting in a NOAEL for calculating avian wildlife values of 2.82×10^{-3} mg/kg/day based on reproductive success.

Most of the avian chronic laboratory studies (presented in Table 1-3) assessed effects of DDT or metabolites on mallard ducks. Numerous accounts of DDT toxicity in birds observed in the field indicate piscivorous birds are among the most severely affected. This is further supported by the acute toxicity data in which the bald eagle is the most sensitive of those tested. It is unlikely, based on the toxicity database, that the pelican is the most sensitive species to the toxicological impacts of DDT and its metabolites; therefore, a SSF of 0.1 is chosen as appropriate.

The wildlife equation and input parameters are presented below.

NOAEL (avian)	=	2.82 µg/kg body weight/day
BAF_3 (Trophic Level 3)	=	1,000,000 ℓ/kg body weight
BAF_4 (Trophic Level 4)	=	3,000,000 ℓ/kg body weight
SSF	=	0.1 (kingfisher, osprey and eagle)

Values for body weights (Wt_A), ingestion rate (F_A), and drinking rate (W_A) for kingfisher, osprey and eagle are presented in Table D-2 of Appendix D to 40 CFR 132, and shown below.

Wt_A (kingfisher)	=	0.15 kg
Wt_A (osprey)	=	1.5 kg
Wt_A (eagle)	=	4.5 kg
F_A (kingfisher)	=	0.075 kg/day
F_A (osprey)	=	0.3 kg/day
F_A (eagle)	=	0.5 kg/day
W_A (kingfisher)	=	0.017 ℓ/day
W_A (osprey)	=	0.077 ℓ/day
W_A (eagle)	=	0.16 ℓ/day

Calculations of avian wildlife values are summarized below.

$$\text{WV (kingfisher)} = \frac{[\text{NOAEL} \times \text{SSF}] \times Wt_{A(\text{kingfisher})}}{W_{A(\text{kingfisher})} + [(1.0)(F_{A(\text{kingfisher})} \times BAF_3)]}$$

$$\text{WV (kingfisher)} = \frac{(2.82\ \mu g/kg/d \times 0.1)\ 0.15\ kg}{0.017\ \ell/d + [(1.0)(0.075\ kg/d \times 1{,}000{,}000\ \ell/kg)]}$$

$$\text{WV (kingfisher)} = 0.56\ pg/\ell$$

$$\text{WV (osprey)} = \frac{[\text{NOAEL} \times \text{SSF}] \times Wt_{A(\text{osprey})}}{W_{A(\text{osprey})} + [(1.0)(F_{A(\text{osprey})} \times BAF_3)]}$$

$$\text{WV (osprey)} = \frac{(2.82\ \mu g/kg/d \times 0.1)\ 1.5\ kg}{0.077\ \ell/d + [(1.0)(0.3\ kg/d \times 1{,}000{,}000\ \ell/kg)]}$$

$$\text{WV (osprey)} = 1.4\ pg/\ell$$

$$\text{WV (eagle)} = \frac{[\text{NOAEL} \times \text{SSF}] \times Wt_{A(\text{eagle})}}{W_{A(\text{eagle})} + [(1.0)(F_{A(\text{eagle})} \times BAF_4)]}$$

$$\text{WV (eagle)} = \frac{(2.82\ \mu g/kg/d \times 0.1)\ 4.5\ kg}{0.16\ \ell/d + [(1.0)(0.5\ kg/d \times 3{,}000{,}000\ \ell/kg)]}$$

$$\text{WV (eagle)} = 0.85\ pg/\ell$$

The geometric mean of these three avian wildlife values results in:

$$WV\ (avian) = e^{((\ln WV(kingfisher) + \ln WV(osprey) + \ln WV(eagle))/3)}$$

$$WV\ (avian) = e^{((\ln 0.58\ pg/\ell + \ln 1.4\ pg/\ell + \ln 0.85\ pg/\ell)/3)}$$

$$WV\ (avian) = 0.87\ pg/\ell.$$

iv. Sensitivity Analysis for Avian Wildlife Value

The values of the various parameters used to derive the avian wildlife value presented above represent the most reasonable assumptions. The purpose of this section is to illustrate the significance of these assumptions and the variability in the avian wildlife value if other assumptions are made for the values of the various parameters from which the avian wildlife value is derived. The intent of this section is to let the risk manager know, as much as possible, the influence on the magnitude of the avian wildlife value of the assumptions made in its derivation.

Anderson et al. (1975) documented significant declines in DDT/DDE levels in the eggs and prey of the brown pelicans, in addition to very moderate declines in the concentrations of PCBs, mercury, and lead in their eggs (Anderson et al., 1977). The presence of these other pollutants in the eggs and prey may have contributed to the observed toxic effects on reproductive success attributed to DDT. However, the levels of these contaminants remained constant over the sampling period and were so low that this was deemed unlikely (Anderson et al., 1975; 1977). Also, throughout the duration of the study, declining DDT and metabolite concentrations were associated with increased eggshell thickness as well as improved reproductive success.

In estimating the hazard of DDT, a SSF value of 0.1 was used to account for possible differences in sensitivity of pelicans compared to kingfisher, osprey, and eagles. This value was based on the fact that these are all piscivorous species and piscivorous species appear more sensitive to the toxicological effects of DDT and its metabolites. If an intermediate SSF of 0.3 (the geometric mean of 1 and 0.1) were used, with the LOAEL determined in the Anderson et al. (1975) study, the avian wildlife value would be of 2.6 pg/ℓ instead of 0.87 pg/ℓ.

The derivation of an avian wildlife value is based on the assumption that 100 percent of an eagle's diet is composed of fish. A study by Kozie and Anderson (1991) suggests that fish comprise 97 percent of Lake Superior eagle diets, and mammals and birds each comprise 1.5 percent of eagle diets. Assuming the metabolizable energy in fish is approximately 1 kcal/g (Palmer, 1988; Stalmaster and Gessaman, 1982) and the typical eagle consumes about 500 g of fish per day (Technical Support Document for Wildlife Criteria, Appendix to the Preamble to 40 CFR 132), an eagle has a daily energy requirement of 500 kcal/day. The energy content for birds is 2 kcal/g (a value derived for mallards; Stalmaster and Gessaman, 1982). Applying the conservative assumptions that the bioaccumulation in mammals would be equivalent to that in Trophic Level 4 fish and the caloric value would be the same for mammals and fish, an eagle diet consisting of 1.5 percent fish-eating birds and 98.5 percent fish would result in a daily intake of approximately 7.4 g of bird and 480 g of fish to meet the daily energy requirement of 500 kcal/day. Braune and Nordstrom (1989) have reported that DDE bioaccummulates in Lake Ontario herring gulls at a level approximately 85 times higher than that observed in alewife. Therefore, dietary exposure of eagles to DDE would be higher if piscivorous birds comprise a portion of their diet. The DDE exposure to eagles eating 7.4 g of piscivorous birds a day would be approximately 2.4 times higher than an exposure associated with a 100 percent fish diet. Such an analysis would result in a bald eagle wildlife value of 0.38 pg/ℓ, and an avian wildlife value of 0.67 pg/ℓ compared to 0.87 pg/ℓ.

IV. Great Lakes Wildlife Criterion

The Tier I Great Lakes Wildlife Criterion for *p,p'*-Dichlorodiphenyltrichloroethane (DDT) and metabolites is determined by the lower of the mammalian wildlife value (270 pg/ℓ) and the avian wildlife value (0.87 pg/ℓ). The avian wildlife value was determined to be approximately 4 orders of magnitude smaller that the mammalian wildlife value. Therefore, the Great Lake Wildlife Criterion for DDT and metabolites is 0.87 pg/ℓ.

i. Discussion of Uncertainties

Wildlife populations inhabiting the Great Lakes Basin would not be impacted from the intake of drinking water or prey taken from surface water containing total DDT in concentrations of 0.87 pg/ℓ, based on available exposure, toxicity and bioaccumulation information, and uncertainty factors applied to account for data gaps and the variability inherent in the DDT risk assessment. Criteria for other ecoregions may require an analysis of different wildlife species with different diets and body masses. In addition, the bioaccumulation factors in this analysis were based on an analysis specific for the Great Lakes; different bioaccumulation factors may be more appropriate for other waterbodies.

Generic assumptions were made in assessing the hazards of DDT and its metabolites to wildlife populations through the use of LOAELs and NOAELs for reproduction and development. The use of these levels assumes no hazards to wildlife populations would result from the direct exposure of individuals to DDT and its metabolites. However, it could be argued that some increase in density independent mortality, or decrease in density independent reproductive success, which could be attributable to exposure to DDT or its metabolites, could be incurred without impacting the population dynamics of a species. In general, well-validated population models do not yet exist for the species analyzed, and it is difficult to estimate the extent of mortality or reproductive failure that could be incurred. In addition, the interaction of additional chemical as well as non-chemical stressors on wildlife population responses is also poorly resolved at this time.

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CHAPTER 2
Tier I Wildlife Criteria for Mercury (Including Methylmercury)

Contents

I. Literature Review 2-1

II. Calculation of Mammalian Wildlife Value 2-1

i. Acute Toxicity Studies 2-1

ii. Chronic Toxicity Studies 2-1

iii. Mammalian Wildlife Value Calculation 2-2

iv. Sensitivity Analysis for Mammalian Wildlife Value 2-3

III. Calculation of Avian Wildlife Value 2-4

i. Acute Toxicity Studies 2-4

ii. Chronic Toxicity Studies 2-5

iii. Avian Wildlife Value Calculation 2-7

iv Sensitivity Analysis for Avian Wildlife Value 2-8

IV. Great Lakes Wildlife Criterion 2-9

i. Discussion of Uncertainties 2-9

V. References 2-9

Tier I Wildlife Criteria for Mercury (Including Methylmercury)

I. Literature Review

A review of the available literature on the environmental cycling, fate, and toxicity of mercury and mercury compounds indicates that criterion derivation for mercury is most appropriately based on methylmercury. A review of mammalian and avian toxicity data for methylmercury was based on literature identified through computer-based (CAS and BIOSES), as well as manual, searches. A total of 27 references were screened; those references which were reviewed in detail are cited in Section V and primarily include those which contained dose-response data.

II. Calculation of Mammalian Wildlife Value

i. Acute Toxicity Studies

Methylmercury and other organomercury compounds are the most toxic forms of mercury to mammals. Methylmercury affects the central nervous system, resulting in sensory, visual, and auditory impairment. Experimentally induced acute mercury poisoning in mule deer (*Odocoileus hemionus*) was characterized by belching, bloody diarrhea, piloerection (i.e., the hair was more erect than usual), and loss of appetite. The kidney appears to be the critical organ affected in adult mammals as a result of the rapid degradation of phenylmercurials and methoxyethylmercurials to inorganic mercury compounds. In the fetus, the brain is the principal target (Eisler, 1987).

The differential toxicity of the different forms of mercury is exemplified by the results of a study by Aulerich et al. (1974). Using adult mink (*Mustela vison*), dietary exposure to 5 ppm of methylmercury was found to be lethal in about 1 month, while exposure to 10 ppm of mercuric chloride did not produce adverse effects over 5 months.

Death in sensitive mammalian species has been associated with daily organomercury doses of 0.1 to 0.5 mg/kg body weight and 1 to 5 mg/kg in the diet. Larger mammals such as mule deer and harp seals (*Pagophilus groenlandicus*) appear to be more resistant to the toxic effects of mercury than smaller mammals. Mule deer had organomercury LD_{50} values of 17.88 mg/kg body weight, and all harp seals exposed to mercury at 25 mg/kg body weight died within 28 days of dietary exposure. Doses of 1.0 mg/kg in the diet produced death in all experimental mink within 2 months of exposure and a dose > 2.0 mg/kg killed all experimental river otters (*Lutra canadensis*); in cats (*Felis domesticus*), convulsions and reductions in survival were associated with organomercury exposure at 0.25 mg/kg body weight for 90 days (Eisler, 1987).

ii. Chronic Toxicity Studies

Wobeser et al. (1976a) examined the effects of organic and inorganic mercury on mink. Wobeser et al. (1976) fed adult female and juvenile ranch mink rations consisting of 50 and 75 percent fish that contained 0.44 ppm total mercury over a 145-day period. The corresponding concentrations of inorganic mercury in the diet are 0.22 and 0.33 ppm. No clinical or pathological signs of intoxication were observed at these exposure concentrations, suggesting a

NOAEL of 0.33 ppm. Using the mink body weight of 1.0 kg and food ingestion rate of 0.15 kg/day provided in the methodology (Appendix D to 40 CFR 132), the NOAEL from this study is 0.05 mg/kg/day.

In a subsequent dose-response study, Wobeser et al. (1976a) fed adult female mink rations treated with methylmercury chloride at concentrations of 1.1, 1.8, 4.8, 8.3, and 15.0 ppm total mercury for 93 days. Mink exposed to dietary mercury concentrations of 1.8 ppm and greater developed signs of clinical intoxication (anorexia, ataxia, and death). The time to onset of the toxic effects was directly related to the mercury content of the ration, and therefore, to the total dose. Pathological alterations in the nervous system were observed at the 1.1 ppm concentration, although they were not associated with any obvious clinical evidence of toxicity. Because these lesions were observed in the nervous systems of animals receiving 1.1 ppm mercury, the authors argued that distinct clinical signs of toxicity would have developed in animals at that dose had the experimental period been longer. Based on these results, the NOAEL for mortality in mink fed methylmercury is 1.1 ppm, and the LOAEL is 1.8 ppm. Using the mink body weight and food ingestion rate presented above, the LOAEL is 0.27 mg/kg/day, and the NOAEL is 0.16 mg/kg/day.

The NOAEL of 0.16 mg/kg/day elemental mercury (methylmercury chloride) reported by Wobeser et al. (1976a) is used to calculate a mammalian-based mercury wildlife value (WV). This study consists of repeated oral exposures for over a 90-day period using a mammalian wildlife species, and therefore meets the criteria for an appropriate study for wildlife criteria development as described in Appendix D to 40 CFR 132.

iii. Mammalian Wildlife Value Calculation

A subchronic to chronic conversion factor of 0.1 is used because of the relatively short duration of the study and the time course of histopathological changes observed in mink fed 0.16 mg/kg/day methylmercury (Wobeser et al. 1976a). This results in an adjusted NOAEL of 0.016 mg/kg/day.

In calculating a Tier I wildlife value, a species sensitivity factor (SSF) within the range of 1 to 0.01 is recommended in Appendix D to 40 CFR 132 to accommodate differences in toxicological sensitivity between the experimental animal and the mink and river otter. A SSF of 1 is used because the NOAEL is based on a study using mink as the test species.

The bioaccumulation factors (BAFs) relate concentration of methylmercury in fish tissue to the concentration of total mercury in the water column. The methylmercury BAFs for Trophic Level 3 and Trophic Level 4 are derived based on the procedure specified in the Great Lakes Water Quality Initiative guidance on bioaccumulation, found in Appendix B to 40 CFR 132, entitled Methodology for Development of Bioaccumulation Factors.

Input parameters for the wildlife equation are presented below.

NOAEL (mammalian)	=	0.016 mg/kg/day
BAF_3(Trophic Level 3)	=	60,000 ℓ/kg body weight
BAF_4(Trophic Level 4)	=	130,000 ℓ/kg body weight
SSF	=	1 (mink and otter)

Wt_A, F_A, and W_A for mink and river otter are presented in Table D-2 of the methodology presented in Appendix D to 40 CFR 132 and are shown below.

Wt_A(mink)	=	1.0 kg
Wt_A(otter)	=	8.0 kg
F_A(mink)	=	0.15 kg/d
F_A(otter)		0.9 kg/d
W_A(mink)		0.099 ℓ/d
W_A(otter)	=	0.64 ℓ/d

The wildlife equations and calculations of mammalian wildlife values are summarized below.

$$WV\ (mink) = \frac{[NOAEL \times SSF] \times Wt_{A(mink)}}{W_{A(mink)} + [(1.0)(F_{A(mink)} \times BAF_3)]}$$

$$WV\ (mink) = \frac{(0.016\ mg/kg/d \times 1)\ 1.0\ kg}{0.099\ \ell/d + [(1.0)(0.15\ kg/d \times 60{,}000\ \ell/kg)]}$$

$$WV\ (mink) = 1{,}800\ pg/\ell$$

$$WV\ (otter) = \frac{[NOAEL \times SSF] \times Wt_{A(otter)}}{W_{A(otter)} + [(0.5)(F_{A(otter)} \times BAF_3) + (0.5)(F_{A(otter)} \times BAF_4)]}$$

$$WV\ (otter) = \frac{(0.016\ mg/kg/d \times 1)\ 8.0\ kg}{0.64\ \ell/d + [(0.5)(0.90\ kg/d \times 60{,}000\ \ell/kg) + (0.5)(0.90\ kg/d \times 130{,}000\ \ell/kg)]}$$

$$WV\ (otter) = 1{,}500\ pg/\ell$$

The geometric mean of these two mammalian wildlife values results in:

$$WV\ (mammalian) = e^{[(\ln WV(mink) + \ln WV(otter))/2]}$$

$$WV\ (mammalian) = e^{[(\ln 1{,}800\ pg/\ell + \ln 1{,}500\ pg/\ell)/2]}$$

$$WV\ (mammalian) = 1{,}600\ pg/\ell.$$

iv. Sensitivity Analysis for Mammalian Wildlife Value

The values of the various parameters used to derive the mammalian wildlife value presented above represent the most reasonable assumptions. The purpose of this section is to illustrate the significance of these assumptions and the variability in the mammalian wildlife value if other assumptions are made for the values of the various parameters from which the mammalian wildlife value is derived. The intent of this section is to let the risk manager know, as much as possible, the influence on the magnitude of the mammalian wildlife value of the assumptions made in its derivation.

In deriving the mammalian wildlife value for mercury, it was assumed that 100 percent of the mink diet was comprised of fish, although this may not necessarily be the case. This assumption may lead to an overestimate of mercury exposure for those organisms that are not

primarily feeding on fish and aquatic invertebrates. However, as indicated in the Technical Support Document for Wildlife Criteria (Appendix to the Preamble to 40 CFR 132), the fish content of a mink diet can vary from less than 50 percent to the 100 percent assumed in the mink wildlife value derivation presented above. If it were assumed only 50 percent of a mink's diet was from aquatic resources and the remaining 50 percent of the diet was uncontaminated, the estimated mercury exposure would be reduced by a factor of 2. The mink wildlife value would be 3600 pg/ℓ, and the mammalian wildlife value would be 2,600 pg/ℓ, rather than the mammalian wildlife value of 1,600 pg/ℓ.

III. Calculation of Avian Wildlife Value

i. Acute Toxicity Studies

Methylmercury has been shown to be more toxic to avian species than inorganic mercury. Acute oral toxicity of methylmercury produced LD_{50} values ranging from 2.2 to 23.5 mg/kg for mallards (*Anas platyrhynchos*), 11.0 to 27.0 mg/kg for quail (*Coturnix*), 14.4 to 33.7 for Japanese quail (*Coturnix japonica*), and 23.8 mg/kg for northern bobwhite (*Colinus virginianus*). Inorganic mercury produced LD_{50} values of 26.0 to 54.0 mg/kg in quail, and 31.1 in Japanese quail (Eisler, 1987). The LD_{50} values for avian species are summarized in Table 2-1. Furthermore, some birds poisoned by inorganic mercury recovered after treatment was withdrawn, while chicks that were fed methylmercury usually died, even after the treated feed was removed.

Mercury poisoning in birds is characterized by muscular incoordination, falling, slowness, fluffed feathers, calmness, withdrawal, hyperactivity, hypoactivity, and eyelid drooping (Eisler, 1987). Following acute oral exposures, signs of mercury poisoning have been observed within 20 minutes after administration in mallards, to 2.5 hours after administration in pheasants. Death occurred between 4 and 48 hours in mallards and 2 and 6 days in pheasants (Hudson et al., 1984).

Table 2-1. Summary of Avian Acute Oral Toxicity Values

Mercury Form	Species	LD_{50} (mg/kg)
Inorganic	Japanese quail (*Coturnix japonica*)	14.1 - 33.7
	Coturnix (*Cotrunix coturnix*)	2956 - 5086
Organic	Chukar (*Alectoris chukar*)	26.9
	Mallard (*Anas platyrhynchos*)	2.2 - 75.7
	Northern bobwhite (*Colinus virginianus*)	23.8
	Coturnix (*Cotrunix coturnix*)	4 - 27
	Japanese quail (*Coturnix japonica*)	14.4 - 33.7
	Rock dove (*Columba livia*)	22.8
	Fulvous whistling duck (*Dendrocygna bicolor*)	37.8
	Domestic chicken (*Gallus domesticus*)	60
	House sparrow (*Passer domesticus*)	12.6 - 37.8
	Gray partridge (*Perdix*)	17.6
	Ring-necked pheasant (*Phasianus colchicus*)	11.5 - 112
	Prairie chicken (*Tympanucus cupido*)	11.5

Source: Eisler (1987).

ii. Chronic Toxicity Studies

Fimreite (1970) raised two-week old leghorn cockerel chicks (*Gallus*) on commercial feed containing methylmercury dicyandiamide at concentrations of 0, 6, 12, and 18 ppm for 21 days. A significant increase in mortality was observed at the highest concentration of methylmercury (18 ppm); however, mortality in chicks maintained at 6 or 12 ppm was not significantly different than that in the control group. Hence, the NOAEL for mortality is 12 ppm. Growth was significantly reduced in chicks maintained on mercury treated feed, suggesting a LOAEL for growth of 6.0 ppm. Using a juvenile chicken body weight of 0.8 kg and a food consumption rate of 0.14 kg/day (NIOSH, 1991) the NOAEL for mortality resulting from ingestion of methylmercury in chicken can be calculated to be 2.1 mg/kg/day, and the LOAEL for growth can be calculated to be 1.1 mg/kg/day.

In another study, Fimreite (1971) exposed ring-necked pheasants to grain treated with a seed dressing containing 2.5 percent methylmercury dicyandiamide at doses of mercury equivalent to approximately 0.69 mg/kg/day, 0.37 mg/kg/day, and 0.18 mg/kg/day for 12 weeks. The laying hens showed no acute symptoms of mercury poisoning; however, adverse effects on reproduction of the pheasants were observed at all dose levels. Reduced hatchability was the most significant effect, while reduced egg production and increased numbers of shell-less eggs were also observed. Among the eggs that hatched, chick mortality appeared to be only slightly increased. The results of this study suggest a LOAEL for total mercury as methylmercury effects on reproduction in pheasants of 0.18 mg/kg/day.

Scott (1977) provided white leghorn laying hens with methylmercury chloride at dietary concentrations of 0, 10, and 20 ppm, and inorganic mercury (mercuric sulfate) at concentrations of 100 and 200 ppm. Methylmercury at 10 and 20 ppm was found to severely impact egg production and weight, fertility of eggs, hatchability of fertile eggs, and eggshell strength. Dietary levels of 100 or 200 ppm inorganic mercury had little affect on egg production, hatchability, shell quality, morbidity, and mortality. The LOAEL for reproductive effects of methylmercury in white leghorn chickens obtained from this study is 10 ppm. Using a chicken body weight of 1.66 kg (Lillie et al., 1975; and personal communication with Dr. Wayne Kunzel, Poultry Science Department, University of Maryland) and a food ingestion rate of 0.81 kg/day derived from the allometric relationship presented in the methodology (Appendix D to 40 CFR 132), the LOAEL for reproductive effects of methylmercury is 4.9 mg/kg/day.

In a series of studies Heinz (1974, 1975, 1976, 1976a, 1979) assessed the effects of dietary methylmercury over three generations of mallard ducks. Adult mallards and ducklings were maintained on a commercial feed treated with methylmercury dicyandiamide at concentrations equivalent to 0, 0.5, and 3.0 ppm elemental mercury (the nominal treatment levels were confirmed by atomic absorption analysis for elemental mercury).

Initially, adult mallard ducks were maintained for a period of up to 21 weeks on the treated diet (Heinz, 1974). There were no consistently large differences in eggshell thickness among the three groups. Egg production stopped earlier among the 3 ppm group than among the 0.5 ppm or control group. Hatching success and hatchling viability, as measured by the number of normal hatchlings and survival of hatchlings through one week, were significantly reduced in the 3.0 ppm group but not in the 0.5 ppm group. These results indicate a LOAEL for reproduction of 3.0 ppm and a NOAEL of 0.5 ppm.

Reproduction in first and second generation ducks was also evaluated (Heinz, 1976, and 1976a). In the first generation, no significant reproductive effects were reported based on an assessment of percent cracked eggs, egg production, or the number of eggs producing normal hatchlings. However, offspring survival to 1-week was significantly lower in the 3.0 ppm treatment group, but not in the 0.5 ppm group. In the second generation of parents fed dietary mercury, abnormal egg-laying behavior, impaired reproduction, and slowed growth of ducklings were observed in the ducks fed 0.5 ppm mercury (Heinz, 1976a). These results suggest a LOAEL of 3.0 ppm and a NOAEL of 0.5 ppm for offspring survival in the first generation, and a LOAEL of 0.5 ppm for reproductive effects in the second generation of offspring.

The reproductive and behavioral effects of methylmercury during the third breeding season are reported in Heinz (1979). During the final year of the study, ducks were only provided with 0 or 0.5 ppm dietary elemental mercury in feed. The results of this study, combined with the earlier investigations (Heinz, 1974, 1975, and 1976) provide dose-response relationships over three generations of mallard ducks. Heinz (1979) found that third generation hens fed methylmercury at 0.5 ppm laid fewer sound eggs than controls. Fewer sound eggs were also observed in the 0.5 ppm group when data were combined across all generations. The percent of incubated eggs producing normal hatchlings and the percent of normal hatchlings surviving 1 week were not significantly reduced by dietary methylmercury exposures. Only during the second generation was the number of 1-week old ducklings produced significantly reduced; however, when pooling these data with the results of the first and third generations, a significant effect was detected.

Heinz (1975, 1976, 1976a, and 1979) examined approach and avoidance in mallard ducklings maintained on treated diets and on the hatchlings born in the second and third breeding season. The behavior tests were designed to measure the approach response to maternal calls, and the avoidance response to a frightening stimulus. Among the initial group of ducklings, alteration of the approach and avoidance behavior was observed at the 0.5 ppm level. During the second generation, the ducklings of parents who were fed 3 ppm mercury were hyper-responsive compared to controls and ducklings from parents fed 0.5 ppm. Altered duckling approach responses were observed in the third generation and when data were pooled over all generations at the low treatment level. Avoidance behavior of ducklings was not significantly altered within any generation. However, when data were pooled across generations, a significant effect was obtained at the low dietary concentration of 0.5 ppm. During the second generation and for the combined data across generations, Heinz (1979) reported that hens laid a significantly higher percentage of eggs outside the nestbox.

Based on the observed adverse reproductive and behavioral effects across the three generations, a LOAEL of 0.5 ppm elemental mercury, as methylmercury, can be inferred. In the multi-generational mallard study, a food consumption rate for mallard ducks was reported to be 128 g/kg/day based on the combined data for controls from the second and third generations. Multiplying the 0.5 ppm dietary mercury LOAEL by the food consumption rate results in a LOAEL of 0.064 mg/kg/d.

The results of the studies described previously are summarized in Table 2-2.

Table 2-2. Summary of Avian Chronic Studies

Species	LOAEL (mg/kg/day)	NOAEL (mg/kg/day)	Toxic Effect Observed	Reference
Chicken (juvenile)	1.1		Growth	Fimreite, 1970
		2.1	Mortality	
Pheasant	0.18		Reproduction	Fimreite, 1971
Chicken	4.9		Reproduction	Scott, 1977
Mallard	0.064		Reproduction, behavior	Heinz, 1974, 1975, 1976, 1976a, 1979

The results of the Heinz (1974, 1975, 1976, 1976a, and 1979) multigeneration studies of the effects of methylmercury on mallard ducks were judged to be the most appropriate for derivation of the avian wildlife value. These studies provide a chemical-specific dose-response curve over three generations with explicitly quantified effects on reproduction and behavior. These effects clearly have potential consequences on populations of mallards exposed to methylmercury.

iii. Avian Wildlife Value Calculation

The LOAEL was divided by a LOAEL to NOAEL uncertainty factor of 2 because the LOAEL appeared to be very near the threshold for dietary effects. Applying this factor to the LOAEL presented previously gives a NOAEL for calculating avian wildlife values of 3.2×10^{-2} mg/kg/day.

Given the limited number of species for which dose-response data is available on the chronic effects of mercury and the lack of avian NOAEL data in these studies, a SSF of 0.1 is used to calculate a wildlife value for kingfisher, osprey, and eagle.

The wildlife equation and input parameters are presented below. The BAFs relate concentration of methylmercury in fish tissue to the concentration of total mercury in the water column.

NOAEL (avian)	=	32 µg/kg body weight/day
BAF_3 (Trophic Level 3)	=	60,000 ℓ/kg body weight
BAF_4 (Trophic Level 4)	=	130,000 ℓ/kg body weight
SSF	=	0.1 (kingfisher, osprey, and eagle)

Values for body weights (Wt_A), food ingestion rate (F_A), and water ingestion rate (W_A) for kingfisher, osprey, and eagle are presented in Table D-2 of the methodology document (Appendix D to 40 CFR 132) and shown below.

Wt_A(kingfisher)		0.15 kg
Wt_A(osprey)		1.5 kg
Wt_A(eagle)		4.5 kg
F_A(kingfisher)		0.075 kg/d
F_A(osprey)		0.3 kg/d
F_A(eagle)	=	0.5 kg/d
W_A(kingfisher)	=	0.017 ℓ/d
W_A(osprey)	=	0.077 ℓ/d
W_A(eagle)	=	0.16 ℓ/d

Calculations of avian wildlife values are summarized below.

$$WV\ (kingfisher) = \frac{(NOAEL \times SSF) \times Wt_{A(kingfisher)} WV\ (eagle)}{W_{A(kingfisher)} + [(1.0)(F_{A(kingfisher)} \times BAF_3)]}$$

$$WV\ (kingfisher) = \frac{(32\ \mu g/kg/d \times 0.1)\ 0.15\ kg}{0.017\ \ell/d + [(1.0)(0.075\ kg/d \times 60{,}000\ \ell/kg)]}$$

$$WV\ (kingfisher) = 100\ pg/\ell$$

$$WV\ (osprey) = \frac{(NOAEL \times SSF) \times Wt_{A(osprey)}}{W_{A(osprey)} + [(1.0)(F_{A(osprey)} \times BAF_3)]}$$

$$WV\ (osprey) = \frac{(32\ \mu g/kg/d \times 0.1)\ 1.5\ kg}{0.077\ \ell/d + [(1.0)(0.3\ kg/d \times 60{,}000\ \ell/kg)]}$$

$$WV\ (osprey) = 260\ pg/\ell$$

$$WV\ (eagle) = \frac{(NOAEL \times SSF) \times Wt_{A(eagle)}}{W_{A(eagle)} + [(1.0)(F_{A(eagle)} \times BAF_4)]}$$

$$WV\ (eagle) = \frac{(32\ \mu g/kg/d \times 0.1)\ 4.5\ kg}{0.16\ \ell/d + [(1.0)(0.5\ kg/d \times 130{,}000\ \ell/kg)]}$$

$$WV\ (eagle) = 220\ pg/\ell$$

The geometric mean of these three avian wildlife values results in

$$\boxed{\begin{aligned} WV\ (avian) &= e^{((\ln WV(kingfisher) + \ln WV(osprey) + \ln WV(eagle))/3)} \\ WV\ (avian) &= e^{((\ln 100\ pg/\ell + \ln 260\ pg/\ell + \ln 220\ pg/\ell)/3)} \\ WV\ (avian) &= 180\ pg/\ell. \end{aligned}}$$

iv. Sensitivity Analysis for Avian Wildlife Value

The values of the various parameters used to derive the avian wildlife value presented above represent the most reasonable assumptions. The purpose of this section is to illustrate the significance of these assumptions and the variability in the avian wildlife value if other assumptions are made for the values of the various parameters from which the avian wildlife value is derived. The intent of this section is to let the risk manager know, as much as possible, the influence on the magnitude of the avian wildlife value of the assumptions made in its derivation.

The derivation of the avian wildlife value is based on the assumption that 100 percent of the eagle's diet is composed of fish. A study by Kozie and Anderson (1991) suggests that fish comprise 97 percent of Lake Superior eagle diets, and mammals and birds each comprise 1.5 percent of eagle diets. Assuming the metabolizable energy in fish is approximately 1 kcal/g (Palmer, 1988; Stalmaster and Gessaman, 1982) and the typical eagle consumes about 500 g of fish per day (Technical Support Document for Wildlife Criteria, Appendix to the Preamble to 40 CFR 132), an eagle has a daily energy requirement of 500 kcal/day. The energy content for birds is 2 kcal/g (a value derived for mallards; Stalmaster and Gessaman, 1982). Applying the

conservative assumptions that the bioaccumulation in mammals would be equivalent to that in Trophic Level 4 fish and the caloric value would be the same for mammals and fish, an eagle diet consisting of 1.5 percent fish-eating birds and 98.5 percent fish would result in a daily intake of approximately 7.4 g of bird and 480 g of fish to meet the daily energy requirement of 500 kcal/day. If methylmercury biomagnifies in fish-eating birds as has been observed for 2,3,7,8-TCDD, DDT, and PCBs (see Braune and Norstrom, 1989), dietary exposure of methylmercury to eagles would be higher if piscivorous birds comprise a portion of their diet than if the diet were composed of 100 percent fish. However, a quantitative estimate of an avian wildlife value adjusted for this additional exposure can not be determined because the empirical data on bioaccumulation of mercury at higher trophic levels is not available.

IV. Great Lakes Wildlife Criterion

The Tier I Great Lakes Wildlife Criterion for mercury is determined by the lower of the mammalian wildlife value (1,600 pg/ℓ) and the avian wildlife value (180 pg/ℓ). The avian wildlife value is one order of magnitude lower than the mammalian value. Therefore the Great Lakes Wildlife Criterion for mercury is 180 pg/ℓ.

i. Discussion of Uncertainties

Wildlife populations inhabiting the Great Lakes Basin would not be impacted from the intake of drinking water or prey taken from surface water containing total mercury in concentrations of 180 pg/ℓ, based on available exposure, toxicity and bioaccumulation information, and uncertainty factors applied to account for data gaps and the variability inherent in the mercury risk assessment. Criteria for other ecoregions may require an analysis of different wildlife species with different diets and body masses than were used for the Great Lakes Basin. In addition, the bioaccumulation factors in this analysis were based on an analysis specific for the Great Lakes; different bioaccumulation factors may be more appropriate for other waterbodies.

Finally, generic assumptions were made in assessing the hazards of mercury to wildlife populations through the use of LOAELs and NOAELs for reproduction and development. The use of these levels assumes no hazards to wildlife populations would result from the direct exposure of individuals to mercury. However, it could be argued that some increase in density independent mortality, or decrease in density independent reproductive success, which could be attributable to mercury exposure, could be incurred without impacting the population dynamics of a species. In general, well-validated population models do not yet exist for the species analyzed, and it is difficult to estimate the extent of mortality or reproductive failure that could be incurred. In addition, the interaction of additional chemical as well as non-chemical stressors on wildlife population responses is also poorly resolved at this time.

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CHAPTER 3
Tier I Wildlife Criteria for 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD)

Contents

I. Literature Review 3-1

I. Calculation of Mammalian Wildlife Value 3-1

i. Acute Toxicity 3-1

ii. Chronic Toxicity 3-2

iii. Mammalian Wildlife Value Calculation 3-4

iv. Sensitivity Analysis for Mammalian Wildlife Value 3-5

III. Calculation of Avian Wildlife Value 3-6

i. Acute Toxicity 3-6

ii. Chronic Toxicity 3-6

iii. Avian Wildlife Value Calculation 3-7

iv. Sensitivity Analysis of Avian Wildlife Value 3-8

IV. Great Lakes Wildlife Criterion 3-9

i. Discussion of Uncertainties 3-9

V. References 3-10

Tier I Wildlife Criteria for 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD)

I. Literature Review

A review of mammalian and avian toxicity data for 2,3,7,8-TCDD was based on literature received through computer-based (CAS and BIOSES) as well as manual searches. A total of 13 references were screened; those references that were reviewed in detail are cited in Section V, and primarily include those that contain dose-response data.

II. Calculation of Mammalian Wildlife Value

i. Acute Toxicity

The toxicity of 2,3,7,8-TCDD to mammals varies greatly both across mammalian species and within a given mammalian species. Large differences between mammalian species exist in the lethal dosages and toxic effects associated with acute doses of 2,3,7,8-TCDD. A difference of more than 8400 fold for LD_{50} values following single oral doses exists between guinea pigs (0.6-2 μg/kg) and hamsters (1157-5051 μg/kg) (Harless et al., 1982; Kociba and Schwetz, 1982). Intraspecific differences in acute responses within a single species have also been observed. For example, LD_{50} values following oral exposure to 2,3,7,8-TCDD have varied from 182 to 2570 μg/kg in three different strains of mice. Rozman (1984) determined the intraperitoneal (i.p.) LD_{50} dose of 2,3,7,8-TCDD to be 60 μg/kg in rats. Mammalian LD_{50} values for 2,3,7,8-TCDD are summarized in Table 3-1.

Acute toxic responses to 2,3,7,8-TCDD by mammals has been characterized by progressive loss of body weight, appetite suppression, and delayed lethality (Eisler, 1986). Rats treated with a single oral dose of 2,3,7,8-TCDD (5, 15, 25, and 50 μg/kg) have displayed a dose-related depression in food intake and body weight (Seefeld and Peterson, 1983). This "wasting syndrome" has been characterized in rats i.p. dosed with 60 μg/kg of 2,3,7,8-TCDD (Rozman, 1984), and rats provided a single oral dose of 50 μg/kg (Seefeld et al., 1984).

Table 3-1. Mammalian Acute Toxicity Values

Route	Species	LD_{50} (μg/kg)
oral	guinea pig	0.6 - 19
oral	rat	22 - 45
oral	Rhesus monkey	< 70
oral	dog	100 - 200
oral	mouse	114 - 2570

Table 3-1. Mammalian Acute Toxicity Values (Cont.)

Route	Species	LD_{50} (μg/kg)
oral	rabbit	115
oral	hamster	1157 - 5051
i.p.	mink (newborn)	1[a]
oral	mink (adult)	4.2[b]

Source: Eisler (1986), except for [a]Aulerich et al. (1988) and [b]Hochstein et al. (1988).

The most sensitive wildlife mammalian species tested was the mink (*Mustela vison*). The i.p. LD_{50} value for 2,3,7,8-TCDD was determined to be 1 μg/kg in newborn mink following 12 days' exposure (Aulerich et al., 1988). The 28-day oral LD_{50} was determined to be 4.2 μg/kg for adult mink (Hochstein et al., 1988).

ii. Chronic Toxicity

No subchronic or chronic studies were obtained for mammalian wildlife species, however, chronic toxicity of 2,3,7,8 TCDD in wildlife species can be extrapolated from results of a number of subchronic and chronic studies using laboratory animals.

Kociba et al. (1978) reported on a two-year toxicity and oncogeny study, using rats (Sprague-Dawley, 50 males and 50 females per group) administered dietary doses of 0, 0.001, 0.01, and 0.1 μg/kg/day. Hematological endpoints, urinary parameters, and gross and microscopic observations on tissues for tumors and tumor-like lesions were evaluated. Animals given the high dose (0.1 μg/kg/day) exhibited increased mortality, decreased weight gain, slight depression of erythroid parameters, increased urinary excretion of porphyrins and delta-aminolevulinic acid and increased serum levels of certain enzymes. Increased tumor incidence and histopathologic or gross effects were observed in liver, lymphoid, lung, and vascular tissues of the high dose animals, and to a lesser extent in the mid-dose group. The liver was the organ that was most consistently affected in rats given 0.1 or 0.01 μg/kg/day, exhibiting multiple hepatocellular degenerative, inflammatory, and necrotic changes. Kociba et al. (1978) concluded that lifetime ingestion of 0.001 μg/kg/day caused no effects considered to be of any toxicological significance. This study, therefore, reported a NOAEL of 0.001 μg/kg/day, and a LOAEL of 0.01 μg/kg/day for liver effects in rats.

Khera and Ruddick (1972) assessed the postnatal effect of prenatal exposure to 2,3,7,8-TCDD. Pregnant Wistar rats were given 0, 0.125, 0.25, 0.5, or 1.0 μg/kg/day TCDD from days 6 through 15 of gestation. Dose-related decreases in the average litter size and pup weight at birth were noted in all but the 0.125 μg/kg/day dose level. Survival of pups to 21 days was significantly reduced at 0.5 μg/kg/day, and there were no surviving pups at 1.0 μg/kg/day. In addition, decreases in the incidence of pregnancy and average litter size were noted in the f_1 generation of the 0.05 μg/kg/day group. These results suggest a NOAEL of 0.125 and a LOAEL of 0.25 μg/kg/day for reproductive effects of TCDD on Wistar rats.

Murray et al. (1979) exposed three generations of Sprague-Dawley rats to dietary 2,3,7,8-TCDD. Rats were maintained on diets equivalent to daily intake rates of 0, 0.001, 0.01, and 0.1 μg/kg/day for at least 90 days prior to gestation and throughout the gestation period. Fertility was significantly reduced among the rats given 0.1 μg/kg/day. At the 0.01 μg/kg/day dose, no effect on fertility was observed among the f_0 rats, but a significant reduction in fertility was observed among the f_1 and f_2 rats. No significant difference was observed between the fertility of the 0.001 μg/kg/day rats and the controls. Significantly decreased litter sizes and gestational survival were

noted among the f_0 0.1 μg/kg/day group and the f_1 and f_2 rats receiving TCDD at 0.01 μg/kg/day. Gestational survival was also significantly reduced among the 0.001 μg/kg/day f_2 generation, but not in earlier or later generations. Significant decreases in postnatal body weight were observed among the f_2 and f_3 litters but not the f_1 litter of the 0.01 μg/kg/day group. Average body weight of pups of rats given 0.1 μg/kg/day, or any generation of the 0.01 μg/kg/day group, were not significantly different from those of control pups. The reproductive capacity of rats in the 0.001 μg/kg/d group was not significantly affected in any generation, but it was reduced in the f_1 and f_2 generations of the 0.01 μg/kg/day group. Therefore, a NOAEL of 0.001 μg/kg/d and a LOAEL of 0.01 μg/kg/d for reproductive capacity of Sprague-Dawley rats were determined from this study.

Bowman et al. (1989, 1989a) reported impaired reproductive success of Rhesus monkeys exposed to 25 parts per trillion (ppt) (0.67 ng/kg/day) but not to 5 ppt (0.13 ng/kg/day) 2,3,7,8-TCDD in feed after 7 and 24 months. The exposures were discontinued after 4 years, and breeding 10 months post-exposures for 4 years did not indicate reproductive impairment. The offspring from these breeding experiments were evaluated for development and behavioral effects (Bowman et al., 1989). While no significant effects of TCDD exposure were found on birth weight, growth, or physical appearance of the offspring, results of some behavioral tests, including alterations in social behavior, were considered to be indicative of TCDD effects. The reproduction study of Bowman et al. (1989a) provides clear evidence of a LOAEL at 0.67 ng/kg/day and a NOAEL at 0.13 ng/kg/day for reproductive effects of TCDD on Rhesus monkeys.

Studies by Schantz et al. (1979), and Allen et al. (1979) also suggest that Rhesus monkeys are more sensitive to 2,3,7,8-TCDD than rats. Decreases in fertility, increased abortions and other toxic effects (e.g., alopecia, hyperkeratosis, weight loss, decreased hematocrit and white blood cell count, and increased serum levels of SGTP) were noted at dietary levels of 50 ppt (1.5 ng/kg/day).

The results of some of the studies described previously are summarized in Table 3-2. The study reported by Murray et al. (1979), in which three generations of Sprague-Dawley rats were exposed to 2,3,7,8-TCDD, was selected for use in developing the mammalian wildlife value (WV). This study was selected because it consists of a multi-generational study that demonstrates a dose-response to 2,3,7,8-TCDD exposure for reproductive effects. The reproduction study by Bowman et al. (1989, 1989a) on Rhesus monkeys was not selected, although a lower NOAEL was determined in this study, because the study by Murray et al. (1979) incorporated a multi-generational exposure regime. In addition, the lack of comparative toxicity endpoints for the mink or river otter and the Rhesus monkey make it very difficult to estimate interspecies uncertainty factors to apply if this study were used.

Table 3-2. Summary of Chronic Mammalian Studies

Species	LOAEL (μg/kg/day)	NOAEL (μg/kg/day)	Toxic Effect Observed	Reference
Rat	0.01	0.001	Liver effects	Kociba et al., 1978
Rat	0.25	0.125	Reproductive	Khera and Ruddick, 1972
Rat	0.01	0.001	Reproductive	Murray et al., 1979
Rhesus Monkey	6.7×10^{-4}	1.3×10^{-4}	Reproductive	Bowman et al., 1989, 1989a

iii. Mammalian Wildlife Value Calculation

In calculating a mammalian wildlife value, a species sensitivity factor (SSF) of 0.1 is used to reflect the uncertainty in extrapolating toxicity data from the rat to the mink and river otter. This SSF value is supported by the limited number of chronic toxicity studies available, the limited number of mammalian species for which chronic data is available, and the extreme sensitivity of mink among those mammalian species for which acute toxicity data is available.

The wildlife equation and input parameters are presented below.

NOAEL (mammalian)	=	0.001 µg/kg body weight/day
BAF_3 (Trophic Level 3)	=	79,000 ℓ/kg body weight
BAF_4 (Trophic Level 4)	=	79,000 ℓ/kg body weight
SSF	=	0.1 (mink and otter)

Body weights (Wt_A), ingestion rates (F_A), and drinking rates (W_A) for mink and river otter are presented in Table D-2 of the methodology document in Appendix D to 40 CFR 132 and shown below.

Wt_A (mink)	=	1.0 kg
Wt_A (otter)	=	8.0 kg
F_A (mink)	=	0.15 kg/day
F_A (otter)	=	0.9 kg/day
W_A (mink)	=	0.099 ℓ/day
W_A (otter)	=	0.64 ℓ/day

The equations and calculations of mammalian wildlife values are presented below.

$$WV\ (mink) = \frac{[NOAEL \times SSF] \times Wt_{A(mink)}}{W_{A(mink)} + [(1.0)(F_{A(mink)} \times BAF_3)]}$$

$$WV\ (mink) = \frac{(0.001\ \mu g/kg/d \times 0.1)\ 1.0\ kg}{0.099\ \ell/d + [(1.0)(0.15\ kg/d)(79{,}000\ \ell/kg)]}$$

$$WV\ (mink) = 8.4 \times 10^{-3}\ pg/\ell$$

$$WV\ (otter) = \frac{[NOAEL \times SSF] \times Wt_{A(otter)}}{W_{A(otter)} + [(0.5)(F_{A(otter)} \times BAF_3) + (0.5)(F_{A(otter)} \times BAF_4)]}$$

$$WV\ (otter) = \frac{(0.001\ \mu g/kg/d \times 0.1)\ 8.0\ kg}{0.64\ \ell/d + [(0.5)(0.90\ kg/d \times 79{,}000\ \ell/kg) + [(0.5)(0.90\ kg/d \times 79{,}000\ \ell/kg)]}$$

$$WV\ (otter) = 1.1 \times 10^{-2}\ pg/\ell$$

The geometric mean of these two mammalian wildlife values results in

$$WV\ (mammalian) = e^{[(\ln WV(mink) + \ln WV(otter))/2]}$$

$$WV\ (mammalian) = e^{[(\ln 0.0084\ pg/\ell + \ln 0.011\ pg/\ell)/2]}$$

$$WV\ (mammalian) = 9.6 \times 10^{-3}\ pg/\ell.$$

iv. Sensitivity Analysis for Mammalian Wildlife Value

The values of the various parameters used to derive the mammalian wildlife value presented above represent the most reasonable assumptions. The purpose of this section is to illustrate the significance of these assumptions and the variability in the mammalian wildlife value if other assumptions are made for the values of the various parameters from which the mammalian wildlife value is derived. The intent of this section is to let the risk manager know, as much as possible, the influence on the magnitude of the mammalian wildlife value of the assumptions made in its derivation.

In deriving the mammalian wildlife value for 2,3,7,8-TCDD, it was assumed that 100 percent of the mink diet was comprised of fish, although this may not necessarily be the case. This assumption may lead to an overestimate of the 2,3,7,8-TCDD exposure for mink that are not primarily feeding on fish and aquatic invertebrates. As indicated in the Technical Support Document for Wildlife Criteria (Appendix to the Preamble to 40 CFR 132), the fish content of a mink diet can vary from less than 50 percent to the 100 percent assumed in the mink wildlife value derivation presented above. If it were assumed only 50 percent of a mink's diet was from aquatic resources and the remaining 50 percent of the diet was uncontaminated, the estimated 2,3,7,8-TCDD exposure would be reduced by a factor of 2. The resulting mink wildlife value would be 1.7×10^{-2} pg/ℓ, and the mammalian wildlife value would be 1.4×10^{-2} pg/ℓ, rather than the mammalian wildlife value of 9.6×10^{-3} pg/ℓ.

As with all criterion derivations, there are uncertainties in assessing risk. The NOAEL derived from Murray et al.(1979) for reproductive effects of 2,3,7,8-TCDD on rats concludes that no adverse effects will be observed at that dose. However, a reevaluation of the Murray et al. (1979) data by Nisbet and Paxton (1982) using different statistical methods (i.e. pooling data from different generations) indicated that both lower dose levels resulted in toxic effects, including significant reductions in offspring survival indices, increases in liver and kidney weight of pups, decreased thymus weight of pups, decreased neonatal weights, and increased incidence of dilated renal pelvis. Nisbet and Paxton (1982) concluded that 0.001 μg/kg/day was a LOAEL and not a NOAEL in the Murray et al. (1979) study. Another evaluation by Kimmel (1988) considered the Murray et al. (1979) data to be suggestive of a pattern of decreased offspring survival and increased offspring renal pathology at 0.001 μg/kg/day, even though the pooling of generations by Nisbet and Paxton (1982) was considered to be biologically inappropriate. Assuming that 0.001 μg/kg/day is a LOAEL, and dividing this LOAEL by a LOAEL to NOAEL uncertainty factor of 10 results in a mammalian wildlife criterion value of 9.6×10^{-4} pg/ℓ instead of the mammalian wildlife value of 9.6×10^{-3} pg/ℓ.

The mammalian assessment assumed that the mink is one of the most sensitive mammalian species to the toxic effects of TCDD, and a SSF value of 0.1 was used to estimate a mink/otter NOAEL from the rat NOAEL. A comparison of toxic effect levels between the Rhesus monkey (Schantz et al., 1979; Allen et al., 1979; Bowman et al., 1989, 1989a) and the rat (Murray et al., 1979) suggests that the monkey may also be quite sensitive. The mammalian wildlife value may be calculated using the Rhesus monkey NOAEL of 0.13 ng/kg/day from the studies by Bowman et al. (1989, 1989a) (a NOAEL value approximately 8 times lower than that determined for the rat). Use of a SSF of 0.1 and the monkey reproductive NOAEL would result in a mammalian wildlife value of 1.3×10^{-3} pg/ℓ. However, in such an analysis, the use of a SSF of 0.1 to predict a mink/otter NOAEL from the result of the Rhesus monkey study may be unduly conservative,

given that the monkey and mink both appear to be extremely sensitive to the toxic effects of 2,3,7,8-TCDD. Use of the Rhesus monkey NOAEL of 0.13 ng/kg/day, and an intermediate SSF of 0.3 would result in a mammalian wildlife value of 3.8×10^{-3} pg/ℓ compared to 9.6×10^{-3} pg/ℓ.

III. Calculation of Avian Wildlife Value

i. Acute Toxicity

Single oral LD_{50} values in avian species for 2,3,7,8-TCDD were reported by Eisler (1986). These LD_{50} values vary from 15 μg/kg in Northern bobwhite quail (*Colinus virginianus*) to more than 810 μg/kg for the ringed turtle dove (*Streptopelia risoria*). Mallards (*Anas platyrhynchos*) are intermediate in sensitivity with an acute oral LD_{50} value of more than 108 μg/kg. Domestic chickens are relatively more sensitive to 2,3,7,8-TCDD than other avian species with an estimated oral LD_{50} range of 25 to 50 μg/kg. The LD_{50} values for avian species are summarized in Table 3-3.

Acute toxic responses to TCDD in birds are characterized by enlarged livers (turtle doves and domestic chickens), emaciation, vomiting, excessive drinking, central nervous system effects (bobwhite quail), and signs of chick edema disease (chickens) (Eisler, 1986).

Table 3-3. Avian Acute Toxicity Values

Species	LD_{50} (μg/kg)
Northern bobwhite quail (*Colinus virginianus*)	15
Ringed turtle-dove (*Streptopelia risoria*)	> 810
Mallard (*Anas platyrhynchos*)	> 108
Domestic chicken (*Gallus domesticus*)	25 - 50

Source: Eisler (1986).

ii. Chronic Toxicity

Environmental mixtures of halogenated aromatic hydrocarbons have been implicated in a number of adverse effects including reproductive failure in avian species (Gilbertson et al., 1991). In such field studies, the observation of reduced reproduction has been correlated to 2,3,7,8-TCDD equivalents; however, the dose-response relationship specific to 2,3,7,8-TCDD itself cannot be discerned from the effects of other contaminants. Most of the laboratory research directed at the determination of dose-response relationships with TCDD has been based on mammalian species, with very little attention given to avian species.

The research of Nosek et al. (1992, 1992a, and 1993) represents the only comprehensive laboratory investigation of the subchronic toxicity and toxicokinetics of 2,3,7,8-TCDD among avian species. Ring-necked pheasants (*Phasianus colchicus*) were dosed weekly, intraperitoneally, for 10 weeks at an equivalent rate of 0.14, 0.014 and 0.0014 μg/kg/day. Egg production was significantly reduced among pheasants from the 0.14 μg/kg/day group, but not in pheasants from the two lowest dose groups when compared to controls. In addition, the 0.14 μg/kg/day dose was associated with a significant increase in mortality of embryos from the fertilized eggs of those hens. The LOAEL for embryo mortality and fertility was 0.14 μg/kg/day; therefore, the NOAEL determined from this study is 0.014 μg/kg/day.

The reproductive effect NOAEL for 2,3,7,8-TCDD determined from the Nosek et al. (1992, 1992a, and 1993) studies will be used in calculating the avian wildlife value. The data generated from this study show a clear dose-response with a meaningful endpoint and are based on exposures lasting more than 28 days. This study is based on an i.p. injection study rather than an oral route of administration. It is generally acknowledged that i.p. and oral routes of exposure are

similar because in both instances the chemical is absorbed first by important internal organs such as the liver, thereby permitting first-pass metabolism. Use of the i.p. dose levels assumes that 2,3,7,8-TCDD bioavailability and absorption from the gastrointestinal tract and the abdominal cavity are not significantly different. To the extent that an i.p. exposure results in higher or lower 2,3,7,8-TCDD absorption than that associated with an oral exposure, the hazards to avian wildlife may be over- or under-estimated.

iii. Avian Wildlife Value Calculation

In calculating a wildlife value for kingfisher, osprey, and eagle, a SSF of 0.1 is used because of the uncertainty in extrapolating data across species given the paucity of chronic toxicity data. In addition, a comparison of results from *in ovo* studies indicate the chicken (Allred and Strange, 1977) is approximately five times more sensitive than the pheasant (Nosek et al. 1992a). Also, a comparison of single-dose LD_{50} values suggests that although the pheasant is among the more sensitive species tested, there are other birds more susceptible to 2,3,7,8-TCDD intoxication (Eisler, 1986; Nosek et al. 1993).

The wildlife equation and input parameters are presented below.

NOAEL (avian)	=	0.014 µg/kg/day
BAF_3 (Trophic Level 3)	=	79,000 ℓ/kg body weight
BAF_4 (Trophic Level 4)	=	79,000 ℓ/kg body weight
SSF	=	0.1 (kingfisher, osprey and eagle)

Values for body weights (Wt_A), ingestion rate (F_A), and drinking rate (W_A) for kingfisher, osprey, and eagle are presented in Table D-2 of the methodology document (Appendix D to 40 CFR 132) and shown below.

Wt_A (kingfisher)		0.15 kg
Wt_A (osprey)		1.5 kg
Wt_A (eagle)		4.5 kg
F_A (kingfisher)		0.075 kg/day
F_A (osprey)		0.3 kg/day
F_A (eagle)	=	0.5 kg/day
W_A (kingfisher)	=	0.017 ℓ/day
W_A (osprey)	=	0.077 ℓ/day
W_A (eagle)	=	0.16 ℓ/day

Calculations of avian wildlife values are summarized below.

$$WV\ (kingfisher) = \frac{[NOAEL \times SSF] \times Wt_{A(kingfisher)}}{W_{A(kingfisher)} + [(1.0)(F_{A(kingfisher)} \times BAF_3)]}$$

$$WV\ (kingfisher) = \frac{(0.014\ \mu g/kg/d \times 0.1)\ 0.15\ kg}{0.017\ \ell/d + [(1.0)(0.075\ kg/d \times 79{,}000\ \ell/kg)]}$$

$$WV\ (kingfisher) = 3.5 \times 10^{-2}\ pg/\ell$$

$$WV\ (osprey) = \frac{[NOAEL \times SSF] \times Wt_{A(osprey)}}{W_{A(osprey)} + [(1.0)(F_{A(osprey)} \times BAF_3)]}$$

$$WV\ (osprey) = \frac{(0.014\ \mu g/kg/d \times 0.1)\ 1.5\ kg}{0.077\ \ell/d + [(1.0)(0.3\ kg/d \times 79{,}000\ \ell/kg)]}$$

$$WV\ (osprey) = 8.9 \times 10^{-2}\ pg/\ell$$

$$WV\ (eagle) = \frac{[NOAEL \times SSF] \times Wt_{A(eagle)}}{W_{A(eagle)} + [(1.0)(F_{A(eagle)} \times BAF_4)]}$$

$$WV\ (eagle) = \frac{(0.014\ \mu g/kg/d \times 0.1)\ 4.5\ kg}{0.16\ \ell/d + [(1.0)(0.5\ kg/d \times 79{,}000\ \ell/kg)]}$$

$$WV\ (eagle) = 1.6 \times 10^{-1}\ pg/\ell$$

The geometric mean of these three avian wildlife values results in

$$\boxed{\begin{aligned} WV\ (avian) &= e^{((\ln WV(kingfisher) + \ln WV(osprey) + \ln WV(eagle))/3)} \\ WV\ (avian) &= e^{((\ln 0.035\ pg/\ell + \ln 0.089\ pg/\ell + \ln 0.16\ pg/\ell)/3)} \\ WV\ (avian) &= 7.9 \times 10^{-2}\ pg/\ell. \end{aligned}}$$

iv. Sensitivity Analysis of Avian Wildlife Value

The values of the various parameters used to derive the avian wildlife value presented above represent the most reasonable assumptions. The purpose of this section is to illustrate the significance of these assumptions and the variability in the avian wildlife value if other assumptions are made for the values of the various parameters from which the avian wildlife value is derived. The intent of this section to let the risk manager know, as much as possible, the influence on the magnitude of the avian wildlife value of the assumptions made in its derivation.

The lack of chronic toxicity data for avian species other than pheasants results in some uncertainty associated with the development of the avian wildlife value. Based on the single exposure acute toxicity data, it could be argued that species such as bobwhite quail, pheasant, and chicken may be among the most sensitive avian species to 2,3,7,8-TCDD. If this were indeed the case, an intermediate SSF value of 0.3 rather than 0.1 may be appropriate. Use of this uncertainty factor would result in an avian wildlife value of 2.4×10^{-1} pg/ℓ instead of 7.9×10^{-2} pg/ℓ.

A subchronic to chronic uncertainty factor in the avian wildlife value calculation was not used, although the study duration was only 10 weeks, because the reported toxicokinetics of 2,3,7,8-TCDD in laying pheasant hens suggest that a significant portion of the dose is excreted (Nosek et al., 1992). However, if a 10-fold subchronic to chronic uncertainty factor were used, the avian criterion would be 7.9×10^{-3} pg/ℓ. If the 10-fold subchronic to chronic uncertainty

factor were used in combination with a SSF value of 0.3, the avian wildlife criterion would be 2.4×10^{-2} pg/ℓ rather than 7.9×10^{-2} pg/ℓ.

The derivation of an avian wildlife value is based on the assumption that 100 percent of an eagle's diet is composed of fish. A study by Kozie and Anderson (1991) suggests that fish comprise 97 percent of Lake Superior eagle diets and mammals and birds each comprise 1.5 percent of eagle diets. Assuming the metabolizable energy in fish is approximately 1 kcal/g (Palmer, 1988; and Stalmaster and Gessaman, 1982) and the typical eagle consumes about 500 g of fish per day (Technical Support Document for Wildlife Criteria, Appendix to the Preamble to 40 CFR 132), an eagle has a daily energy requirement of 500 kcal/day. The energy content for birds is 2 kcal/g (a value derived for mallards; Stalmaster and Gessaman, 1982). Applying the conservative assumptions that the bioaccumulation in mammals would be equivalent to that in Trophic Level 4 fish and the caloric value would be the same for mammals and fish, an eagle diet consisting of 1.5 percent fish-eating birds and 98.5 percent fish would result in a daily intake of approximately 7.4 g of bird and 480 g of fish to meet the daily energy requirement of 500 kcal/day. Braune and Nordstrom (1989) have reported that 2,3,7,8-TCDD bioaccummulates in Lake Ontario herring gulls at a level approximately 30 times higher than that observed in alewife. Therefore, dietary exposure to eagles of 2,3,7,8-TCDD would be higher if piscivorous birds comprise a portion of their diets. The 2,3,7,8-TCDD exposure to eagles eating 7.4 g of piscivorous birds a day would be approximately 1.4 times higher than an exposure associated with a 100 percent fish diet. Such an analysis would result in a bald eagle wildlife value of 1.1×10^{-1} pg/ℓ, and an avian wildlife value of 7.1×10^{-2} pg/ℓ compared to 7.9×10^{-2} pg/ℓ.

IV. Great Lakes Wildlife Criterion

The Great Lakes Wildlife Criterion for 2,3,7,8-TCDD is determined by the lower of the mammalian wildlife value (9.6×10^{-3} pg/ℓ) and the avian wildlife value (7.9×10^{-2} pg/ℓ). The mammalian wildlife value was determined to be approximately one order of magnitude smaller than the avian wildlife value. Therefore, the Great Lakes Wildlife Criterion for 2,3,7,8-TCDD is 9.6×10^{-3} pg/ℓ.

i. Discussion of Uncertainties

Wildlife populations inhabiting the Great Lakes Basin would not be impacted from the intake of drinking water or aquatic prey taken from surface water containing 2,3,7,8-TCDD in concentrations of 9.6×10^{-3} pg/ℓ, based on the uncertainty factors used to account for data gaps and the variability in the toxicity and exposure parameters inherent in the 2,3,7,8-TCDD risk assessment. Criteria for other ecoregions may require an analysis of different wildlife species with different diets and body masses. In addition, the bioaccumulation factors in this analysis were based on an analysis for the Great Lakes, and different bioaccumulation factors may be more appropriate for other waterbodies.

Finally, generic assumptions were made in assessing the hazards of 2,3,7,8-TCDD to wildlife populations through the use of LOAELs and NOAELs for reproduction and development. The use of these levels assumes no hazards to wildlife populations would result from the direct exposure of individuals to 2,3,7,8-TCDD. However, it could be argued that some increase in density independent mortality, or decrease in density independent reproductive success, which could be attributable to 2,3,7,8-TCDD exposure could be incurred without impacting the population dynamics of a species. In general, well-validated population models do not yet exist for the species analyzed, and it is difficult to estimate the extent of mortality or reproductive failure that could be incurred. In addition, the interaction of additional chemical as well as non-chemical stressors on wildlife population responses is also poorly resolved at this time.

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CHAPTER 4
Tier I Wildlife Criteria for Polychlorinated Biphenyls (PCBs)

Contents

I. Literature Review 4-1

II. Calculation of Mammalian Wildlife Value 4-1

i. Acute Toxicity 4-1

ii. Chronic Toxicity 4-1

iii. Mammalian Wildlife Value Calculation 4-5

iv. Sensitivity Analysis for Mammalian Wildlife Value 4-7

III. Calculation of Avian Wildlife Value 4-7

i. Acute Toxicity 4-7

ii. Chronic Toxicity 4-8

iii. Avian Wildlife Value Calculation 4-11

iv. Sensitivity Analysis of Avian Wildlife Value 4-12

IV. Great Lakes Wildlife Criterion 4-13

i. Discussion of Uncertainties 4-13

V. References 4-14

Tier I Wildlife Criteria for Polychlorinated Biphenyls (PCBs)

I. Literature Review

A review of mammalian and avian toxicity data for polychlorinated biphenyls was based on literature received through computer-based (CAS and BIOSES) as well as manual searches. A total of 41 references were screened; those references which were reviewed in detail are cited in Section V and primarily include those that contain dose-response data.

II. Calculation of Mammalian Wildlife Value

i. Acute Toxicity

Three primary effects of PCB exposure on terrestrial wildlife are mortality, decreased reproductive success, and behavioral modifications. Mink (*Mustela vison*) appear to be among the more sensitive species to the toxic effects of PCBs (Gillette et al., 1987). Single oral doses of PCBs administered to mink have produced LD_{50} values of 750 mg/kg for Aroclor 1221 and 4000 mg/kg for Aroclor 1254 (Aulerich and Ringer, 1977; Ringer, 1983). Diets containing PCBs at 6.7 mg (Aroclor 1254)/kg to 8.6 mg (Aroclor 1242)/kg fresh weight have caused 50 percent mortality among mink over a 9-month period (Ringer, 1983). The reasons for mink sensitivity to PCBs are unknown, but interspecific variability in sensitivity to PCBs is common, even among closely-related species. For example, the acute toxicity (i.e., LC_{50}) of Aroclor 1242 has been demonstrated to be lower among European ferrets (*Mustela putorius furo*) ($LC_{50} > 20$ mg/kg) than among mink ($LC_{50} = 8.6$ mg/kg) (Eisler, 1986). Age, dietary composition, season, and year have had little effect on the outcome of the acute toxicity tests. Acute toxicity values for mink fed a diet containing a mixture of PCBs produced 28- and 35-day LD_{50} values of 79 and 48.5 ppm, respectively (Hornshaw et al., 1986). Acute toxicity values for mink fed a diet consisting of cattle that had been fed Aroclor 1254 were 47 and 31.5 ppm, respectively for 28-day and 35-day LC_{50} tests (Aulerich et al., 1986). In a longer term study, high daily intake of PCBs (Clophen A-60, equivalent to Aroclor 1060) fed to female mink for 51 days caused 100 percent mortality at 6.1 mg/day and 40 percent mortality at 2.0 mg/day (den Boer, 1984). Table 4-1 provides a summary of values of acute mammalian toxicity to PCBs.

ii. Chronic Toxicity

Numerous studies (Ringer et al., 1972; Platonow and Karstad, 1973; Jensen et al. 1977; Aulerich and Ringer, 1977; U.S. EPA, 1980; Bleavins et al. 1980) have demonstrated that mink are among the most sensitive mammalian species to the toxic effects of PCBs, with some PCB congeners being more toxic than others. The main chronic effect that has been documented as a result of dietary exposure to PCBs has been decreased reproductive success, as evidenced by reduced whelping rates, fetal death, and reduced growth among the young.

Table 4-1. Mammalian Acute Toxicity Values

PCB Congener	Route	Species	LD_{50} (g/kg)
1221	oral	rat	1 - 4
	oral	mink	0.75 - 1
	dermal	rabbit	4
	i.p.	mink	0.5 - 0.75
1242	diet	mink	0.0086
	diet	ferret	0.02
	oral	rat	0.8 - 8.7
	oral	mink	3
	dermal	rabbit	8.7
	i.p.	mink	1
1254	metabolized diet	mink	0.0032 - 0.0047[a]
	diet	mink	0.0067
	diet	mouse	> 0.1 - > 0.25
	diet	rat	> 0.075
	diet	raccoon	> 0.05
	diet	rabbit	> 0.01
	oral	rat	0.5 - 1.4
	oral	mink	4
	i.p.	mink	1.25 - 2.25
1260	oral	rat	1.3 - 10
	dermal	rabbit	10

Source: Eisler (1986), except for [a]Aulerich et al. (1986).

Bleavins et al. (1980) investigated the effects of dietary exposure to Aroclors 1016 and 1242 on mink and ferrets. Mink were fed a diet supplemented with either 0, 5, 10, 20, or 40 ppm Aroclor 1242 or 20 ppm Aroclor 1016. The ferrets were fed a diet supplemented with either 0, or 20 ppm Aroclor 1242 or 20 ppm Aroclor 1016 for 9 months. Aroclor 1242 produced 100 percent mortality in all adult mink fed diets at the 20 ppm and 40 ppm levels. Only one female and no males died when fed a diet containing 5 ppm Aroclor 1242. No mortality was noted among mink fed diets containing 20 ppm Aroclor 1016. Mink fed Aroclor 1242 at 5 and 10 ppm failed to reproduce, while Aroclor 1016 reduced but did not completely eliminate reproduction. In contrast to these results, no mortality attributed to the PCBs was observed among the ferrets. Ferrets fed the Aroclor 1242 diet did not whelp, but reproductive performance among the female ferrets fed Aroclor 1016 was not significantly different from that of the control females. In addition, reproductive parameters, kit growth, and adult and kit mortality were not significantly affected in the ferrets fed the Aroclor 1016 diet. Using a mink body weight of 1 kg, and a food consumption rate of 0.15 kg/day, provided in the methodology document (Appendix D to 40 CFR 132), the results from this study suggest a mink reproductive LOAEL of 0.75 mg/kg/day (5 ppm) for Aroclor 1242 and 3.0 mg/kg/day (20 ppm) for Aroclor 1016. The body weights and food

consumption rates of ferrets are virtually identical to mink (Ringer et al., 1981). Using a ferret body weight of 1 kg and a food consumption rate of 0.15 kg/day, the LOAEL for reproductive effects for Aroclor 1242 and the NOAEL for reproductive effects for Aroclor 1016 are 3.0 20.096 mg/mg/kg/day.

Linzey (1988) evaluated reproductive success and growth among white footed mice (*Peromyscus leucopus*) chronically exposed to Aroclor 1254 in the diet at a level of 10 ppm. PCB-treated second generation mice exhibited poor reproductive success in comparison with second generation controls and the parental generation. This was evidenced by reduced reproductive organ weights, drastically reduced number of litters, and survival among the young of the second generation treated group. Poor growth among the second generation PCB-treated litter was also observed, with increasing differences in body weights becoming apparent over time when compared to controls. Using a mouse body weight of 32 grams, and an ingestion rate of 4.9 g/day (U.S. EPA, 1988), the dietary PCB exposure associated with reproductive effects was calculated to be 1.53 mg/kg/day.

According to Platonow and Karstad (1973) and Hornshaw et al. (1983), reproductive impairment occurs in mink at even lower concentrations when the PCBs fed to the mink have first been metabolized by another species. Platonow and Karstad (1973) orally dosed Aroclor 1254 to Jersey cows, and fed the resulting contaminated beef to mink over 160 days at 0.64 and 3.57 ppm total PCBs. At a dietary concentration 3.57 ppm total PCBs, no live kits were produced and all adult mink died before the end of the experiment. At 0.64 ppm total PCBs in the diet, 2 of 14 adult mink died before the end of the experiment and only 1 of 12 mink produced kits. All 3 of the kits died during the first day after birth. Based on these findings the LOAEL for successful reproduction was 0.64 ppm. Based on the mink body weight and food consumption rate presented above, the LOAEL was calculated as 0.096 mg/kg/day for reproductive effects of total PCBs.

Hornshaw et al. (1983) fed Great Lakes fish or fish products to mink for up to 290 days. Dietary concentrations of PCB residues were determined to range from 0.21 to 1.50 ppm. Only mink fed PCBs at concentrations of 0.21 ppm had reproduction and kit survival similar to the controls. Mink fed a diet containing 0.48 ppm of PCB residues had inferior reproductive performance and/or kit survival when compared to controls. These findings suggest a NOAEL of 0.21 ppm and a LOAEL of 0.48 ppm. Using the mink body weight and food ingestion rate presented above, the NOAEL was calculated to be 0.032 mg/kg/day, and the LOAEL was 0.072 mg/kg/day for reproductive performance and kit survival. Hornshaw et al. (1983) observed that the toxicity of PCBs was greater when derived from Great Lakes fish than in previous studies using comparable levels of technical grade PCBs. However, concentrations of other toxicants potentially present in the Great Lakes fish were not measured.

Fetotoxicity and reproductive failure have also been reported for mink following direct dietary exposure to low levels of certain PCB congeners. Wren et al. (1987) fed adult ranch-bred mink diets containing either 0 or 1.0 ppm Aroclor 1254, 1.0 ppm methylmercury, a combination of 1.0 ppm Aroclor 1254 and 1.0 ppm methylmercury, or a combination of 0.5 ppm Aroclor 1254 and 0.5 ppm methylmercury. Fertility of adult male mink, percentage of females whelped, or number of kits born per female were not affected by the treatments, but the growth rate of the kits nursed by the mothers exposed to 1.0 ppm Aroclor 1254 (0.15 mg/kg/day) was significantly reduced.

In a sub-chronic study Jensen et al. (1977) dosed mink with PCBs (congeners not reported) in the feed at concentrations of 0.05, 3.3, and 11 ppm for 66 days. Complete reproductive failure was observed among the 11 ppm group, with reduced number of implantation sites and no kits born. The frequencies of mated and pregnant females did not differ significantly between the 0.05 ppm group and the 3.3 ppm dose group. At 3.3 ppm, however, the frequency of delivering females, and the number of kits born per female were significantly smaller than at 0.05 ppm. In addition, the number of stillbirths at 3.3 ppm was greater and the average body weight of the young was smaller than among the controls. From these results, a LOAEL for reproduction can be inferred of 3.3 ppm, and a NOAEL of 0.05 ppm. Using the mink body weight and ingestion

rates presented previously, the LOAEL is calculated as 0.5 mg/kg/day, and the NOAEL is 0.008 mg/kg/day.

Aulerich and Ringer (1977) exposed mink to dietary Aroclor 1254 at 0, 5, and 10 ppm over a 9-month period. All of the mink fed PCB-supplemented diets failed to produce offspring. In a subsequent experiment, mink were provided diets containing 2 ppm Aroclor 1016, 1221, 1242, or 1254, and monitored over 297 days. Aroclor 1254 was the only PCB mixture that had an adverse effect on reproduction. Two of the 7 females whelped and 1 live, underweight kit was produced. Based on these studies, a LOAEL for reproductive success of 2 ppm Aroclor 1254 can be inferred. Using the mink body weight and food consumption rates presented above, a LOAEL was calculated to be 0.3 mg/kg/d for reproductive effects of Aroclor 1254.

Aulerich and Ringer (U.S. EPA, 1980) investigated the effects of Aroclor 1016 on reproduction, growth, and survival of mink. Mink were fed diets that contained 0, 2, 10, and 25 ppm Aroclor 1016 for up to 18 months. No marked hematological changes or clinical signs of PCB poisoning were observed in even the highest dose group; however, increased heart and decreased kidney weights were noted in some animals, but these were not consistent among the treated groups. Reproduction was not adversely affected, but reduced 4-week weights were observed among kits nursed by females fed the 25 ppm PCB supplemented diet, and excessive kit mortality between birth and 4 weeks was noted among most of the groups provided with PCB supplemented diets. The authors attributed these adverse effects to quantitative or qualitative impacts of PCBs on lactation. From these results, a LOAEL of 2 ppm for kit survival and growth can be inferred. Using the mink body weight and feeding rate presented above, this LOAEL is equivalent to 0.3 mg/kg/day.

Aulerich et al. (1985) fed Aroclor 1254 and three hexachlorobiphenyl congeners (2,4,5,2',4',5'- [245 HCB]; 2,3,6,2',3',6'- [236 HCB]; and 3,4,5,3',4',5'- [345 HCB]) to adult female mink for 12.5 weeks at concentrations ranging from 0.1 ppm to 5.0 ppm in the diet (each congener was not given at each dose level). Concentrations of 5 and 2.5 ppm of 245 HCB or 236 HCB had no significant effect on the number of females that whelped or the litter size per female whelped. Only 1 out of 10 females whelped and no live kits were produced at 2.5 ppm Aroclor 1254 in the diet. At 0.5 ppm 345 HCB in the diet, all animals died after 29 to 72 days exposure. At 0.1 ppm 345 HCB in the diet, 50 percent mortality was observed before the end of the experiment and none of the 8 females whelped. Based on the results of Aulerich et al. (1985), a LOAEL for survival and for reproductive effects of 0.1 ppm 345 HCB can be inferred. Using the body weight and food ingestion rate provided above, this LOAEL is equivalent to 0.015 mg/kg/day for survival and reproductive effects of 345 HCB. The LOAEL from this study for reproductive effects of Aroclor 1254 is 0.375 mg/kg/day.

den Boer (1984) investigated reproductive effects of dietary exposure to PCBs originating from fish livers and Clophen A-60 (equivalent to Aroclor 1260) during 400 days. Mink were maintained on feed contaminated with total PCBs at levels equivalent to 25.2 μg/kg/day. No mortality was observed among the dosed groups; however, a significant reduction in females whelping was observed among the exposed mink.

The various toxicity values derived from the studies that were discussed previously are summarized in Table 4-2. An evaluation of these studies suggests that the LOAEL of 0.3 mg/kg/d for reproductive effects of Aroclor 1254, from the study of Aulerich and Ringer (1977), is the most appropriate daily dose rate to use in calculating a mammalian wildlife value (WV) for total PCBs. The LOAEL values for mink developed for HCBs in the Aulerich et al. (1985) study are lower than the LOAEL for Aroclor 1254; however, they cannot be used for criteria development because of a lack of dose-response data. Furthermore, use of the LOAEL for HCB would be based on the unreasonable assumption that all PCBs discharged into the environment are HCBs, or that all the discharged PCBs would be totally converted to 3,4,5-HCB. The LOAELs derived using metabolized PCBs (Platonow and Karstad, 1973; Hornshaw et al., 1983) are not appropriate for criteria development, in part because possible contamination of feed by additional environmental contaminants was not investigated. Therefore, the results of Aulerich and Ringer

(1977) were considered to more properly reflect the toxicity of total PCBs to which mammalian wildlife species are exposed.

iii. Mammalian Wildlife Value Calculation

The LOAEL derived from Aulerich and Ringer (1977) of 0.3 mg/kg/d was based on a 297-day feeding study. This LOAEL was divided by a subchronic to chronic uncertainty factor of 10, resulting in an adjusted LOAEL of 0.03 mg/kg/d. This uncertainty factor was used based on the results of the study by den Boer (1984) in which a significant reduction in females whelping was observed in mink exposed to PCB-treated feed at a concentration of 0.025 mg/kg/day for 400 days, which corresponds to an intake rate approximately 10 times lower than the LOAEL derived from Aulerich and Ringer (1977). As discussed by den Boer (1984), the results of these two studies illustrate that the total amount of PCB intake, rather than the daily dose, is critical in assessing adverse effects. Lower dietary PCB concentrations can cause significant adverse effects with a sufficiently long exposure duration.

A NOAEL for reproductive effects in mink from total PCBs was determined by dividing the LOAEL by a LOAEL to NOAEL uncertainty factor of 10. Thus, the NOAEL used in calculating mammalian wildlife values was 3.0 μg/kg/d.

Table 4-2. Summary of Chronic Mammalian PCB Studies

Species	LOAEL (mg/kg/day)	NOAEL (mg/kg/day)	PCB Congener	Toxic Effect Observed	Reference
Mouse	1.53		Aroclor-1254	Reproductive	Linzey, 1988
Mink	0.75		Aroclor-1242	Reproductive	Bleavins et al. 1980
	3.0		Aroclor-1016		
Ferret	3.0		Aroclor-1242	Reproductive	Bleavins et al. 1980
		3.0	Aroclor-1016		
Mink	0.096		Metabolized Aroclor-1254	Reproductive	Platonow and Karstad, 1973
Mink	0.072	0.032	Metabolized total PCBs	Reproductive/Kit survival	Hornshaw et al. 1983
Mink	0.5		Unreported PCBs	Reproductive	Jensen et al. 1977
Mink	0.15		Aroclor-1254	Kit growth	Wren et al. 1987
Mink	0.3		Aroclor 1254	Reproductive	Aulerich and Ringer, 1977
		0.3	Aroclor-1016		
		0.3	Aroclor-1021		
		0.3	Aroclor-1242		
Mink	0.3		Aroclor-1016	Kit growth	U.S. EPA, 1980
Mink	0.375		Aroclor-1254	Reproductive	Aulerich et al., 1985
		0.375	245 HCB		
		0.375	236 HCB		
	0.015		345 HCB		
Mink	0.025		Clophen A-60	Reproductive	den Boer, 1984

In calculating a wildlife value for both mink and river otter, a species sensitivity factor (SSF) of 1 was used because numerous studies (Ringer et al., 1972; Platonow and Karstad, 1973; Jensen et al. 1977; Aulerich and Ringer, 1977; U.S. EPA, 1980; Bleavins et al. 1980) have demonstrated that mink are among the most sensitive mammalian species to the toxic effects of PCBs.

Input parameters for the wildlife equation are presented below.

NOAEL (mammalian)	=	3.0 µg/kg body weight/day
BAF_3 (Trophic Level 3)	=	1,000,000 ℓ/kg body weight
BAF_4 (Trophic Level 4)	=	2,800,000 ℓ/kg body weight
SSF	=	1 (mink and otter)

Body weights (Wt_A), ingestion rates (F_A), and drinking rates (W_A) for mink and river otter are presented in Table D-2 of the methodology document (Appendix D to 40 CFR 132) and shown below.

Wt_A (mink)		1.0 kg
Wt_A (otter)		8.0 kg
F_A (mink)	=	0.15 kg/day
F_A (otter)	=	0.9 kg/day
W_A (mink)	=	0.099 ℓ/day
W_A (otter)	=	0.64 ℓ/day

The equations and calculations of mammalian wildlife values are presented below.

$$\text{WV (mink)} = \frac{[\text{NOAEL} \times \text{SSF}] \times Wt_{A(mink)}}{W_{A(mink)} + [(1.0)(F_{A(mink)} \times BAF_3)]}$$

$$\text{WV (mink)} = \frac{(3.0\ \mu g/kg/d \times 1)\ 1.0\ kg}{0.099\ \ell/d + [(1.0)(0.15\ kg/d \times 1{,}000{,}000\ \ell/kg)]}$$

$$\text{WV (mink)} = 20\ pg/\ell$$

$$\text{WV (otter)} = \frac{[\text{NOAEL} \times \text{SSF}] \times Wt_{A(otter)}}{W_{A(otter)} + [(0.5)(F_{A(otter)} \times BAF_3) + (0.5)(F_{A(otter)} \times BAF_4)]}$$

$$\text{WV (otter)} = \frac{(3.0\ \mu g/kg/d \times 1)\ 8.0\ kg}{0.64\ \ell/d + [(0.5)\ (0.90\ kg/d \times 1{,}000{,}000\ \ell/kg) + [(0.5)\ (0.90\ kg/d \times 2{,}800{,}000\ \ell/kg)}$$

$$\text{WV (otter)} = 14\ pg/\ell$$

The geometric mean of these two mammalian wildlife values results in

$$\text{WV (mammalian)} = e^{[\ln \text{WV(mink)} + \ln \text{WV(other)}]/2}$$

$$\text{WV (mammalian)} = e^{[\ln 20\ \text{pg}/\ell + \ln 14\ \text{pg}/\ell]/2}$$

$$\text{WV (mammalian)} = 17\ \text{pg}/\ell.$$

iv. Sensitivity Analysis for Mammalian Wildlife Value

The values of the various parameters used to derive the mammalian wildlife value presented above represent the most reasonable assumptions. The purpose of this section is to illustrate the significance of these assumptions and the variability in the mammalian wildlife value if other assumptions are made for the values of the various parameters from which the mammalian wildlife value is derived. The intent of this section to let the risk manager know, as much as possible, the influence on the magnitude of the mammalian wildlife value of the assumptions made in its derivation.

In deriving the PCB mammalian wildlife value, it was assumed that 100 percent of the mink diet was comprised of fish, although this may not necessarily be the case. This assumption may lead to an overestimate of PCB exposure for mink that are not primarily foraging for fish and aquatic invertebrates. As indicated in the Technical Support Document for Wildlife Criteria (Appendix to the Preamble to 40 CFR 132), the fish content of a mink diet can vary from less than 50 percent to the 100 percent assumed in the mink wildlife value derivation presented above. If it were assumed only 50 percent of a mink's diet was from aquatic resources and the remaining 50 percent of the diet was uncontaminated, the estimated PCB exposure would be reduced by a factor of 2. The resulting wildlife value for the mink would be 40 pg/ℓ, and the mammalian wildlife value would be 24 pg/ℓ, rather than the mammalian wildlife value of 17 pg/ℓ.

III. Calculation of Avian Wildlife Value

i. Acute Toxicity

Birds have been shown to be more resistant than mammalian species to the acute toxic effects of PCBs. PCB doses greater than 200 ppm in the diet (10 mg/kg body weight) caused some mortality among northern bobwhite (*Colinus virginians*), mallards (*Anas platyrhynchos*), and ring-necked pheasants (*Phasianus colchicus*). PCBs provided to these birds at dietary concentrations of 1500 ppm (100 mg/kg body weight) have caused extensive mortality (Eisler, 1986). Exposure to PCBs has caused some mortality among all the avian species tested, with lethal concentrations depending on the length of exposure and the particular PCB mixture (Aulerich et al., 1973). Values of LD_{50} for various avian species provided with dietary concentrations of PCBs have varied from 604 mg/kg for the northern bobwhite to more than 6000 mg/kg for the Japanese quail (*Coturnix japonica*) (Heath et al., 1972), while mallards had LC_{50} values of more than 2000 mg/kg (NAS, 1979). Acute toxicity values for avian species are summarized in Table 4-3.

Table 4-3. Avian Acute Toxicity Values

PCB Congener	Species	LD_{50} (mg/kg)
1221	Northern bobwhite (*Colinus virginianus*)	> 6000
	Ring-necked pheasant (*Phasianus colchicus*)	> 4000
	Japanese quail (*Coturnix japonica*)	> 6000
1242	Northern bobwhite (*Colinus virginianus*)	2098
	Mallard (*Anas platyrhynchos*)	> 2000 - 3182

Table 4-3. Avian Acute Toxicity Values (Cont.)

PCB Congener	Species	LD_{50} (mg/kg)
1242 (Cont.)	Ring-necked pheasant (*Phasianus colchicus*)	2078
	Japanese quail (*Coturnix japonica*)	> 6000
1254	Northern bobwhite (*Colinus virginianus*)	604
	Mallard (*Anas platyrhynchos*)	> 2000 - 2699
	Ring-necked pheasant (*Phasianus colchicus*)	1091
	Japanese quail (*Coturnix japonica*)	2898
	European starling (*Sturnus vulgaris*)	1500
	Red-winged blackbird (*Agelaius phoeniceus*)	1500
	Brown-headed cowbird (*Molothrus ater*)	1500
1260	Northern bobwhite (*Colinus virginianus*)	747
	Mallard (*Anas platyrhynchos*)	1975 - > 2000
	Ring-necked pheasant (*Phasianus colchicus*)	1260
	Japanese quail (*Coturnix japonica*)	2186

Source: Eisler (1986).

For all avian species, PCB residue concentrations of at least 310 mg/kg fresh weight in the brain were associated with an increased likelihood of death from PCB poisoning (Eisler, 1986). Residues in brains of starlings (*Sturnus vulgaris*), red-winged blackbirds (*Agelaius phoeniceus*), common grackles (*Quiscalus quiscula*), and brown-headed cowbirds (*Molothrus ater*) that died after ingesting diets containing 1500 ppm of Aroclor 1254 ranged from 349 to 763 mg/kg. Brains of birds surviving at the 50 percent mortality point contained 54 to 301 ppm PCBs (Stickel et al. 1984).

ii. Chronic Toxicity

Chronic toxicity studies have been conducted on mallards, Japanese quail, pheasants, and domestic leghorn chickens (*Gallus*). Chickens have been shown to be more sensitive to the effects of chronic exposure to PCBs than have other avian species.

Custer and Heinz (1980) fed 9-month-old mallards with a dietary dosage of 25 ppm Aroclor 1254 for at least a month before egg-laying. Treatment did not affect reproductive success or nest attentiveness during incubation. The number of hens laying, date of the first egg laid, clutch size, survival of ducklings to 3 weeks of age, and the number of times off the nest per day and total time off the nest per day did not differ between the exposed group and the controls. Fertility of eggs was greater among the treated birds than among controls, a phenomenon that the authors attributed to males coming into reproductive condition sooner as a result of the PCBs. Using a mallard body weight of 1 kg (Delnicki and Reinecke, 1986) and food ingestion rate of 0.058 kg/day derived from the allometric equation provided in the methodology document (Appendix D to 40 CFR 132), the treatment concentration can be calculated to be equivalent to a dose of 1.45 mg/kg/day.

In contrast to the results of the mallard study, dietary exposure to PCBs had marked effects among chickens at the same or lower concentrations. Britton and Huston (1973) exposed white leghorn hens to Aroclor 1242 at 0, 5, 10, 20, 40, and 80 ppm in a commercial feed over a 6-week period. Following treatment, the hens were held for an additional 6 weeks on a PCB-free

diet and effects on reproduction were assessed. Dietary PCBs did not alter egg weight, shell thickness, or shell weight over the 12-week experiment. PCBs in the diet did have an effect on the hatchability of eggs. By the second week, no eggs laid by hens fed 80 ppm PCBs hatched. Hatchability improved as the concentration of PCBs in the diet decreased. A significant reduction in hatchability of the eggs laid by hens fed 10 ppm Aroclor 1242 was observed at the sixth week of the experiment, but no effect on hatchability was noted for the eggs laid by hens fed a 5 ppm diet. Using a chicken weight of 1.66 kg (Lillie et al. 1975; and personal communication with Dr. Wayne Kunzel, Poultry Science Department, University of Maryland) and a food ingestion rate of 0.81 kg/day derived from the allometric relationship presented in the methodology document (Appendix D to 40 CFR 132), the NOAEL for Aroclor 1242 determined from this study was calculated to be 2.44 mg/kg/day (5 ppm) for hatchability of eggs.

Aroclor 1254 was also found to cause reduced egg production and hatchability in chickens. In a subchronic study, Platonow and Reinhart (1973) fed chickens rations containing 0, 5, or 50 ppm Aroclor 1254 for up to 39 weeks. A drastic decline in production and hatchability of fertile eggs was observed among hens maintained at the 50 ppm level. At 5 ppm, egg production was reduced, but not the hatchability of the fertile eggs. Fertility for the 5 ppm group was similar to the control during the first 14 weeks, but declined significantly in the last 14 weeks. These results indicate a LOAEL of 5 ppm for egg production and fertility. Using the chicken body weight and feed ingestion rate presented above, the LOAEL was calculated to be 2.44 mg/kg/day.

Lillie et al. (1975) assessed the reproductive effects of various PCBs (i.e., Aroclors 1232, 1242, 1248, 1254, and 1016) on white leghorn chickens maintained on a commercial feed treated at 0, 2, 5, 10, and 20 ppm total PCBs for 8 to 9 weeks. The data presented by Lillie et al. (1975) were pooled, both across Aroclors and across dose rates, making their interpretation unreliable. However, the data indicate no effect on egg production from dietary exposure at any concentration of any of the Aroclors. Furthermore, the data indicate that PCB levels of 5 ppm in feed, regardless of congener, has no effect on hatchability, while Aroclors 1232, 1242 and 1248, regardless of concentration, but probably at 10 and 20 ppm, caused reduced hatchability. None of the Aroclors or dose levels had any effect on egg weight, eggshell thickness, adult body weight changes, feed consumption, livability, or fertility.

In another paper Lillie et al. (1974) found that dietary exposure to either 2 or 20 ppm of the various PCBs had no effect on adult body weight, adult mortality, fertility, egg weight, or eggshell thickness. Reduced egg production and egg hatchability were observed among the different groups of chickens maintained on 20 ppm Aroclor 1232, 1242, 1248, or 1254. These effects were not observed at a dietary concentration of 2 ppm. Lillie et al. (1974) also monitored the growth and survival of chicks produced from hens maintained on Aroclor-treated feed. A significant reduction in growth was observed among chicks produced from hens maintained on feed treated with either Aroclor 1248 or Aroclor 1254 at 2.0 and 20 ppm. Significant reduction in weight gain was also observed among chicks produced from hens maintained on feed treated with either Aroclor 1232 or Aroclor 1242 at 20 ppm but not at 2 ppm. Only Aroclor 1248 at a concentration of 20 ppm in the maternal diet was associated with significant chick mortality. The results of this study indicate a 2.0 ppm NOAEL and a 20 ppm LOAEL for egg production and hatchability with Aroclors 1232, 1242, 1248, or 1254. In addition, a 2.0 ppm LOAEL for chick growth effects for Aroclor 1248 and 1254, and a 2.0 ppm NOAEL for Aroclors 1232 and 1242 can be inferred. Using the chicken body weight and food ingestion rates presented previously, the LOAEL and NOAEL for egg production and hatchability can be calculated to be 9.8 and 0.98 mg/kg/day, respectively. For chick growth effects, the LOAEL for Aroclors 1248 and 1254, and the NOAEL for Aroclors 1232 and 1242 are 0.98 mg/kg/day.

Scott (1977) measured the effect of Aroclor 1248 on reproductive parameters of white leghorn hens maintained at dietary concentrations of 0.5, 1.0, 10.0, and 20.0 ppm over an 8-week period. A significant reduction in egg production at the 20 ppm concentration and a decrease in hatchability of fertile eggs at the 10 ppm dose after 8 weeks were noted. No significant effects on these reproductive endpoints were observed at 1 ppm Aroclor 1248 in the

diet. Using the chicken body weight and food ingestion rate presented above, the NOAEL for reproduction is 0.49 mg/kg/day, and the LOAEL is 4.9 mg/kg/day.

Dahlgren et al. (1972) assessed the effects of orally-administered Aroclor 1254 on reproduction in the ring-necked pheasant. Female pheasants were individually dosed once per week, via gelatin capsule, at rates of 0, 12.5, and 50 mg/week; and male pheasants were dosed at rates of 0 and 25 mg/week, for 16 weeks. Egg production, egg fertility, egg hatchability, survivability, and growth of chicks through 6 weeks post-hatch were monitored. Egg production and chick survivability were significantly reduced among hens administered 50 mg Aroclor 1254 per week, but not among hens administered 12.5 mg per week. No effect of Aroclor 1254 administration on egg fertility was noted, although significant reductions in hatchability were reported among eggs from both treatment groups. No effect of Aroclor treatment on chick growth was observed. Using a pheasant body weight of 1 kg (John Nosek, personal communication), a value of 1.8 mg/kg/day can be inferred from this study for the NOAEL for egg production and chick survivability, and for the LOAEL for egg hatchability.

The various toxicity values derived from the studies discussed above are summarized in Table 4-4. An evaluation of these studies suggest that the lowest LOAEL values are those for chick growth from chickens dosed with Aroclors 1248 and 1254 (Lillie et al. 1974) and the value for egg hatchability among pheasants obtained with Aroclor 1254 (Dahlgren et al. 1972). The lowest NOAELs were for egg production and hatchability among chickens using Aroclors 1232, 1242, 1248, or 1254 (Lillie et al. 1974; Scott, 1977).

The results of the pheasant study by Dahlgren et al. (1972) are used to derive the avian wildlife value. According to the methodology document, preference is given to laboratory studies with wildlife species. Pheasants have been show to be as sensitive to PCBs as chickens, the more traditional avian laboratory species. The toxic endpoint of egg hatchability is a meaningful reproductive effect that is associated with avian dietary exposure to PCBs. In addition, the study by Dahlgren et al. (1972) involved exposures to both male and female adults. Calculation of the avian wildlife values for PCBs is based on the study of Dahlgren et al. (1972), where a LOAEL of 1.8 mg/kg/day for egg hatchability was determined for Aroclor 1254.

Table 4-4. Summary of Chronic Avian PCB Studies

Species	LOAEL (mg/kg/day)	NOAEL (mg/kg/day)	PCB Congener	Toxic Effect Observed	Reference
Mallard		1.45	Aroclor 1254	Reproduction	Custer and Heinz, 1980
Chicken		2.44	Aroclor 1242	Egg hatchability	Britton and Huston, 1973
Chicken	2.44		Aroclor 1254	Egg production and Fertility	Platonow and Reinhart, 1973
Chicken	4.88	2.44	Aroclor 1232	Egg hatchability	Lillie et al. 1975
	4.88	2.44	Aroclor 1242		
	4.88	2.44	Aroclor 1248		
		2.44	Aroclor 1254		
		2.44	Aroclor 1016		
Chicken	9.8	0.98	Aroclor 1232	Egg production and Hatchability	Lillie et al. 1974
	9.8	0.98	Aroclor 1242		

Table 4-4. Summary of Chronic Avian PCB Studies (Cont.)

Species	LOAEL (mg/kg/day)	NOAEL (mg/kg/day)	PCB Congener	Toxic Effect Observed	Reference
Chicken (Cont.)	9.8	0.98	Aroclor 1248	Egg production and Hatchability	Lillie et al. 1974
	9.8	0.98	Aroclor 1254		
		0.98	Aroclor 1232	Chick growth	
		0.98	Aroclor 1242		
	0.98		Aroclor 1248		
	0.98		Aroclor 1254		
Chicken	4.9	0.49	Aroclor 1248	Egg production and Hatchability	Scott, 1977
Pheasant	1.8		Aroclor 1254	Egg hatchability	Dahlgren et al. 1972

iii. Avian Wildlife Value Calculation

Dividing the LOAEL for egg hatchability by a LOAEL to NOAEL uncertainty factor of 10 gives a NOAEL for calculating avian wildlife values of 0.18 mg/kg/day.

Results of the chicken and pheasant studies suggest that these 2 species are similarly sensitive to the toxic effects of PCBs, which suggests that a 0.1 SSF may be unduly conservative in deriving avian-specific wildlife values. In that piscivorous species may be more sensitive to PCB toxicity than the chicken or pheasant, a SSF of 0.3, intermediate to 0.1 and 1.0, was selected.

The wildlife equation and input parameters are presented below.

NOAEL (avian)	=	0.18 mg/kg body weight/day
BAF_3 (Trophic Level 3)	=	1,000,000 ℓ/kg body weight
BAF_4 (Trophic Level 4)	=	2,800,000 ℓ/kg body weight
SSF	=	0.3 (kingfisher, osprey and eagle)

Values for body weights (Wt_A), ingestion rate (F_A), and drinking rate (W_A) for kingfisher, osprey and eagle are presented in Table D-2 of the methodology document (Appendix D to 40 CFR 132), and shown below.

Wt_A (kingfisher)		0.15 kg
Wt_A (osprey)		1.5 kg
Wt_A (eagle)		4.5 kg
F_A (kingfisher)		0.075 kg/day
F_A (osprey)		0.3 kg/day
F_A (eagle)	=	0.5 kg/day
W_A (kingfisher)	=	0.017 ℓ/day
W_A (osprey)	=	0.077 ℓ/day
W_A (eagle)	=	0.16 ℓ/day

Calculations of avian wildlife values are summarized below.

$$WV\ (kingfisher) = \frac{(NOAEL \times SSF) \times Wt_{A(kingfisher)}}{W_{A(kingfisher)} + [(1.0)(F_{A(kingfisher)} \times BAF_3)]}$$

$$WV\ (kingfisher) = \frac{(0.18\ mg/kg/d \times 0.3)\ 0.15\ kg}{0.017\ \ell/d + [(1.0)(0.075\ kg/d \times 1{,}000{,}000\ \ell/kg)]}$$

$$WV\ (kingfisher) = 110\ pg/\ell$$

$$WV\ (osprey) = \frac{(NOAEL \times SSF) \times Wt_{A(osprey)}}{W_{A(osprey)} + [(1.0)(F_{A(osprey)} \times BAF_3)]}$$

$$WV\ (osprey) = \frac{(0.18\ mg/kg/d \times 0.3)\ 1.5\ kg}{0.077\ \ell/d + [(1.0)(0.3\ kg/d \times 1{,}000{,}000\ \ell/kg)]}$$

$$WV\ (osprey) = 270\ pg/\ell$$

$$WV\ (eagle) = \frac{(NOAEL \times SSF) \times Wt_{A(eagle)}}{W_{A(eagle)} + [(1.0)\ (F_{A\ eagle} \times BAF_4)}$$

$$WV\ (eagle) = \frac{(0.18\ mg/kg/d \times 0.3)\ 4.5\ kg}{0.16\ \ell/d + [(1.0)(0.5\ kg/d \times 2{,}800{,}000\ \ell/kg)]}$$

$$WV\ (eagle) = 170\ pg/\ell$$

The geometric mean of these three avian wildlife values results in:

$$WV\ (avian) = e^{((\ln WV(kingfisher) + \ln WV(osprey) + \ln WV(eagle))/3)}$$

$$WV\ (avian) = e^{((\ln 110\ pg/\ell + \ln 270\ pg/\ell + \ln 170\ pg/\ell)/3)}$$

$$WV\ (avian) = 170\ pg/\ell.$$

iv. Sensitivity Analysis of Avian Wildlife Value

The values of the various parameters used to derive the avian wildlife value presented above represent the most reasonable assumptions. The purpose of this section is to illustrate the significance of these assumptions and the variability in the avian wildlife value if other assumptions are made for the values of the various parameters from which the avian wildlife value is derived. The intent of this section is to let the risk manager know, as much as possible, the influence on the magnitude of the avian wildlife value of the assumptions made in its derivation.

No chronic PCB toxicity studies using piscivorous avian species were identified; however, it could be assumed that such species are more sensitive to the effects of PCBs than the 0.3 SSF would suggest. Use of a SSF of 0.1 would result in an avian wildlife value of 57 pg/ℓ instead of 170 pg/ℓ.

Chickens have been shown to be among the most sensitive species to PCB toxicity. Chronic toxicity studies with chickens suggest effects on reproductive success could be expected at a threshold between 0.24 and 0.98 mg/kg/day (Lillie et al. 1974, 1975). Using these values as the NOAEL in calculating avian wildlife values, and using a SSF of 0.3 yields avian wildlife values of 230 pg/ℓ to 940 pg/ℓ, respectively. The use of a SSF of 0.1 would result in avian wildlife values ranging from 76 pg/ℓ to 310 pg/ℓ instead of the avian wildlife value of 170 pg/ℓ.

Mallard studies are also available to calculate wildlife values, and these may be considered more representative of sensitive wildlife species than those from chicken or pheasant. Mallard studies yield a NOAEL for reproduction of 1.45 mg/kg/day (Custer and Heinz, 1980). If the results of the mallard study were used with a SSF of 0.3, the avian wildlife value would be approximately 1400 pg/ℓ. If the mallard NOAEL were used with a SSF of 0.1, the avian wildlife value would be approximately 460 pg/ℓ, instead of the avian wildlife value of 170 pg/ℓ.

The derivation of an avian wildlife value is based on the assumption that 100 percent of an eagle's diet is composed of fish. A study by Kozie and Anderson (1991) suggests that fish comprise 97 percent of Lake Superior eagle diets, and mammals and birds each comprise 1.5 percent of eagle diets. Assuming the metabolizable energy in fish is approximately 1 kcal/g (Palmer, 1988; and Stalmaster and Gessaman, 1982) and the typical eagle consumes about 500 g of fish per day (Technical Support Document for Wildlife Criteria, Appendix to the Preamble to 40 CFR 132), an eagle has a daily energy requirement of 500 kcal/day. The energy content for birds is 2 kcal/g (a value derived for mallards; Stalmaster and Gessaman, 1982). Applying the conservative assumptions that the bioaccumulation in mammals would be equivalent to that in Trophic Level 4 fish and the caloric value would be the same for mammals and fish, an eagle diet consisting of 1.5 percent fish-eating birds and 98.5 percent fish would result in a daily intake of approximately 7.4 g of bird and 480 g of fish to meet the daily energy requirement of 500 kcal/day. Braune and Nordstrom (1989) have reported that total PCBs bioaccummulate in Lake Ontario herring gulls at a level approximately 90 times higher than that observed in alewife. Therefore, dietary exposure to eagles of total PCBs would be higher if piscivorous birds comprise a portion of their diets. The total PCBs exposure to eagles eating 7.4 g of piscivorous birds a day would be approximately 2.3 times higher than an exposure associated with a 100 percent fish diet. Such an analysis would result in a bald eagle wildlife value of 75 pg/ℓ, and an avian wildlife value of 130 pg/ℓ compared to 170 pg/ℓ.

IV. Great Lakes Wildlife Criterion

The Great Lake Wildlife Criterion for polychlorinated biphenyls (PCBs) is determined by the lower of the mammalian wildlife value (17 pg/ℓ) and the avian wildlife value (170 pg/ℓ). The mammalian wildlife value is one order of magnitude smaller than the avian wildlife value. Therefore, the Great Lake Wildlife Criterion for polychlorinated biphenyls (PCBs) is 17 pg/ℓ.

i. Discussion of Uncertainties

Wildlife populations inhabiting the Great Lakes basin would not be impacted from the intake of drinking water and aquatic prey taken from surface water containing PCBs in concentrations of 17 pg/ℓ, based on the uncertainty factors used to account for data gaps and the variability in the toxicity and exposure parameters inherent in the PCB risk assessment. Criteria for other ecoregions may require an analysis of different wildlife species with different diets and body masses. In addition, the bioaccumulation factors in this analysis were based on an analysis for the Great Lakes, and different bioaccumulation factors may be more appropriate for other waterbodies.

Finally, generic assumptions were made in assessing the hazards of PCBs to wildlife populations through the use of LOAELs and NOAELs for reproduction and development. The use of these levels assumes no hazards to wildlife populations would result from the direct exposure of individuals to PCBs. However, it could be argued that some increase in density independent mortality, or decrease in density independent reproductive success, which could be attributable to exposure to PCBs, could be incurred without impacting the population dynamics of a species. In general, well-validated population models do not yet exist for the species analyzed, and it is difficult to estimate the extent of mortality or reproductive failure that could be incurred. In addition, the interaction of additional chemical as well as non-chemical stressors on wildlife population responses is also poorly resolved at this time.

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